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The Surgical Correction of Calcific Aortic Stenosis in Adults

I. Technique of Transaortic Valvuloplasty*

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THE SURGICAL CORRECTION of calcific aortic stenosis in adults, particularly those in congestive failure, constitutes a great surgical challenge. The many operative techniques currently in use, including open direct visualization under hypothermia[1,2] or cardiopulmonary bypass,[3–5] transventricular fracture-dilatation with valvulotomes[6–10] and various retrograde transaortic operations,[11–13] suggest that one optimal approach has not been developed.

After experience with various open and closed techniques our present procedure can be summarized as follows. Open transaortic operation with cardiac bypass (not hypothermia) is used in noncalcific and subaortic stenosis. The closed Ivalon® tunnel technique described here is employed in heavily calcific aortic stenosis in adults. When there is doubt as to calcification, or when a rubbery stenotic funnel might compromise the closed technique, the closed technique is used with pump-oxygenator ready for open transaortic operation as needed. There remains little place for blind transventricular valvulotome operations, no matter how simple they are technically. Similarly we hold no place for hypothermia alone in these procedures. Of the transaortic operative techniques the Ivalon operating tunnel has specific advantages over cloth, pericardium, blood vessels, and rubber. The reasons for this preference are presented subsequently here and in another publication.[14]

In some parts of the world repeated clinical disasters have led to the conviction that efforts to correct calcific aortic stenosis in adults should now be abandoned. It is the purpose of this report to correct this impression by describing a supra-aortic approach that has been used with increasing success in 100 consecutive patients. We will consider certain aspects of the life cycle, pathologic morphology, clinical and hemodynamic features of aortic stenosis

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which have a bearing on the selection of patients for surgical relief. Further details of hemodynamic evaluation and clinical follow-up are the subjects of further communications.[14,20] Finally, the limitations of any technique which aims at restoration of normal valve function in these badly damaged valves encourage further efforts in the development of prosthetic valves.[15–17]

The Disease

The natural history of aortic stenosis differs significantly from that of mitral or tricuspid stenosis.[18] Congenital, noncalcific aortic stenosis in children will not be included in this discussion. Doubtless some calcific aortic stenosis in adults is congenital in origin, but it is similar hemodynamically, clinically, and surgically to adult calcific rheumatic and arteriosclerotic forms. The differentiation of etiology may at times be difficult, even on most careful pathologic examination.

Clinical Aspects: The murmur of aortic stenosis generally precedes disability by many years.[19] The average age of onset of symptoms is later in this disease than in mitral stenosis, and many patients reach the fifth or sixth decade before experiencing difficulty. Others complete a normal life expectancy if the narrowing does not enter the critical range. On the other hand, calcific disease is not uncommon in the third decade, and a history of a murmur since childhood suggests that this lesion is congenital. The clinical course following the onset of symptoms is similar to that found in older patients with acquired forms of aortic stenosis.

It has been repeatedly observed that the dyspnea associated with mitral stenosis may seriously limit a patient for as long as five or even ten years, and yet death not be imminent.[20] This is true because the lungs are burdened early and directly by the obstructive back pressure and apparently have effective mechanisms of compensation. In contrast, in the patient with aortic stenosis dyspnea develops only when the left ventricle begins to fail, and from this point his life expectancy is markedly shortened.

Pathophysiology: The underlying physiologic derangement in aortic stenosis depends on two factors.[21] First, the peripheral leaflet sclerosis in arteriosclerotic marginal leaflet fusion may cause a loud murmur or murmurs long before there is any hemodynamic disturbance. Second, even after there is valve impedance there may be a long symptom-free period before rapid deterioration and death. The congestive failure which heralds the onset of the terminal phase may be due to the discrepancy between the muscle mass of the left ventricle and its blood supply. In the absence of coronary arterial narrowing the left ventricle may remain compensated for a long time before it outgrows its blood supply.

Because of the large work capacity of the left ventricle and its ability to overcome orificial resistance by marked increase in intraventricular pressure, "critical narrowing" does not appear until a valve size of approximately 0.5 sq. cm is reached.[22,23] This is in sharp contrast to the "critical value" of 1.0 sq. cm in mitral stenosis.[24] As the muscle mass of the left ventricle enlarges in response to the increased work imposed by valvular narrowing, further increments of output in response to effort become progressively smaller until the stroke output and the work are fixed. At this point the coronary blood supply is barely able to nourish the burdened myocardium. Even a slight further demand may then throw the left ventricle into deficit or failure that is characteristically intractable since all compensating factors capable of supporting the vital coronary circulation have been exhausted. This "forward failure" has much more serious implications than the "backward failure" of mitral stenosis, for the viability of the heart itself is at stake.

In a patient with auricular fibrillation in addition to aortic stenosis, the decompensating point occurs much earlier because of the added burden of inefficient rhythm.[18]

Hemodynamic Features: Gorlin and colleagues[22] prepared a useful diagram that expresses the relationships between valve area, aortic valve flow and pressure gradient (Fig. 1). The inner arc crossing the others indicates the pressure-flow relations which usually exist at different valve areas in pure stenosis. They add that "because of the prodigious demands of left ventricular systolic pressure, the stroke output tends not to rise above a certain point and to be fixed at proportionately lower levels, the greater the stenosis." They describe two further fundamental hemodynamic features of mixed aortic stenosis and insufficiency.

First, when aortic insufficiency is added to stenosis, the extra volume of blood that must be ejected adds sharply to the ventricular work. A large amount of blood can reflux during

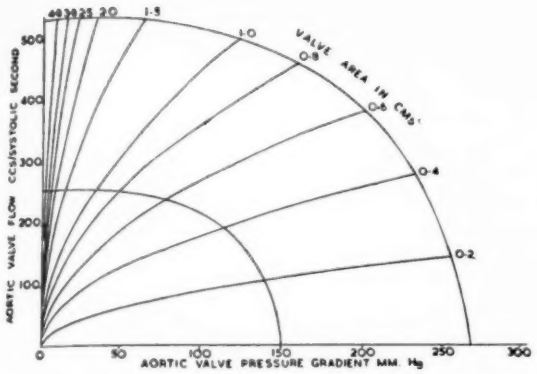

FIG. 1. "Gorlin fan" shows relation between pressure and flow in various degrees of aortic stenosis. Note minimal increase in flow even with marked increase in gradient when valve area is 0.5 sq. cm or less. From: GORLIN, R. *et al.* Dynamics of the circulation in aortic valvular disease. *Am. J. Med.*, 18: 855, 1955.

diastole because of the large aorticoventricular diastolic gradient and all this is a still greater burden in systole because of the stenosis. *Second*, and conversely, when there is sufficient associated mitral stenosis to reduce cardiac output, the left ventricular pressure necessary to maintain such a reduced output may be within the physiologic range; thus, left ventricular overload may not occur.

Prognosis: The classic triad of symptoms in aorticstenosis, angina, syncope and left ventricular failure, is well known. Levine,[19] White,[25] and others have emphasized that once any of these appear, the course may be inexorable and even accelerated. More recently, Bergeron *et al.*[18] have indicated that "within two years of the advent of any of these symptoms one-half of the patients had died. Less than a quarter of the patients survived the onset of these manifestations by five years." In their experience when auricular fibrillation was added the deterioration was much more rapid and only 25 per cent of such patients survived one year. Ellis also believes that auricular fibrillation is just as ominous a development as left ventricular failure. Finally, he and his colleagues comment that "when either (congestive failure or auricular fibrillation) was accompanied by cardiac pain, death followed within weeks to months."

This unfavorable prognosis is further emphasized by our repeated experience of recommending operation for patients with symptomatic aortic stenosis only to have them die of the disease before reaching the hospital. Fifty-four such patients were urged to have an operation but for various reasons did not accept or come to operation. Of these 54 patients, who constitute an unplanned "control series," 49 died within six months. These cases have impressed us with the urgency of surgical relief of aortic stenosis as soon as it becomes symptomatic. It is, of course, possible that this series is weighted because the patients have not been referred to us in many instances until they were literally terminal, and therefore died before surgery. In others, apprehension may have delayed acceptance of the physician's advice until some alarming episode, such as a pulmonary embolus, prompted a hurried consultation. Nevertheless, in spite of these factors we must still conclude that it is "later than one thinks" in symptomatic aortic stenosis. The good general clinical appearance of the patient can be very misleading.

This "control" experience contrasts with the encouraging operative results to be discussed. The recent improvement in operative mortality and quality of aortic valvuloplasty may warrant relaxation in our indications for surgical intervention. The former requirement of at least slight left ventricular failure as manifested by dyspnea now may be dangerously severe.

DIAGNOSIS OF AORTIC STENOSIS

The evaluation of cardiac valvular lesions has extended beyond murmur identification to include hemodynamic appraisal. As effective surgical correction of specific lesions has developed, it has always necessitated an improvement in diagnostic accuracy to avoid unnecessary operations. The poor correlation between the loudness of murmur and significance of the valvular lesion has been mentioned.[18,23] In the evaluation of aortic stenosis the myocardial blood supply must also be taken into consideration. Early in our experience we learned to recognize that the patient with the murmur of aortic stenosis and angina or left ventricular failure, but with a normal-sized heart, might well be limited more by coronary artery disease than his valvular lesion.

In addition to the characteristic "diamond-shaped murmur" in the aortic area transmitted to the neck, we look for radiologic evidence of left ventricular hypertrophy. The electrocardiogram, which is undoubtedly more sensitive than the roentgenogram, often shows left ventricular enlargement in both coronary artery disease and pure aortic stenosis although it is

more marked in the latter. The diagnosis of aortic stenosis in the absence of this finding on the electrocardiogram is rarely justified.

Differentiation of Aortic Stenosis and Insufficiency: In the presence of both systolic and diastolic aortic murmurs hemodynamic evaluation becomes more complicated. Pure aortic insufficiency, because of roughening of the valve leaflets or adherence of cusps at the commissures, is almost invariably associated with a rough systolic murmur even though there is no significant narrowing of the valve orifice. Both lesions produce left ventricular hypertrophy on the roentgenogram and electrocardiogram. Significant aortic insufficiency generally produces a diastolic pressure of less than 60 and a pulse pressure of more than 50 mm Hg. However, Gorlin and Goodale[26] have emphasized that the diastolic blood pressure in aortic insufficiency may rise as the patient goes into left ventricular failure, and again fall with compensation. Thus, one must not overemphasize the diastolic pressure in the presence of congestive failure.

One of the most useful means of differentiating aortic stenosis from insufficiency is to observe the left ventricle at fluoroscopy. In pure aortic stenosis the hypertrophied left ventricle shows little excursion and there is reduced pulsation in the ascending aorta. There is often conspicuous poststenotic dilatation beyond the calcified valve. With careful positioning one can often visualize the specific calcified cusps and determine which are mobile. It is generally possible to differentiate punctuate, bicuspid, or tricuspid stenotic lesions in this way. By contrast, in pure aortic insufficiency a "rocking beat" occurs. This is due to the large volume of systolic ejection and reflux, forming a wave from ventricle to aorta and back. The enlarged left ventricle shows a wide excursion alternating with increased aortic expansion and subsequent "run-off."

When arteriosclerotic heart disease, aortic insufficiency or associated mitral valvular disease are present, it may be even more difficult to determine the relative importance of the various valvular lesions. Similarly, when the clinical history is less than clear, other methods of hemodynamic appraisal are necessary. Here left heart catheterization combined with indicator-dilution studies[27-29] represents a very useful tool. We have used a simple modification of Fisher's percutaneous technique (Fig. 2).

Morphology of Aortic Stenosis

The conventional method of studying the pathologic anatomy of the heart (i.e., opening the chambers and valve rings in the direction of blood flow and subsequently preserving this mass of tissue in stiffening and decolorizing solutions) seems diabolically contrived to con-

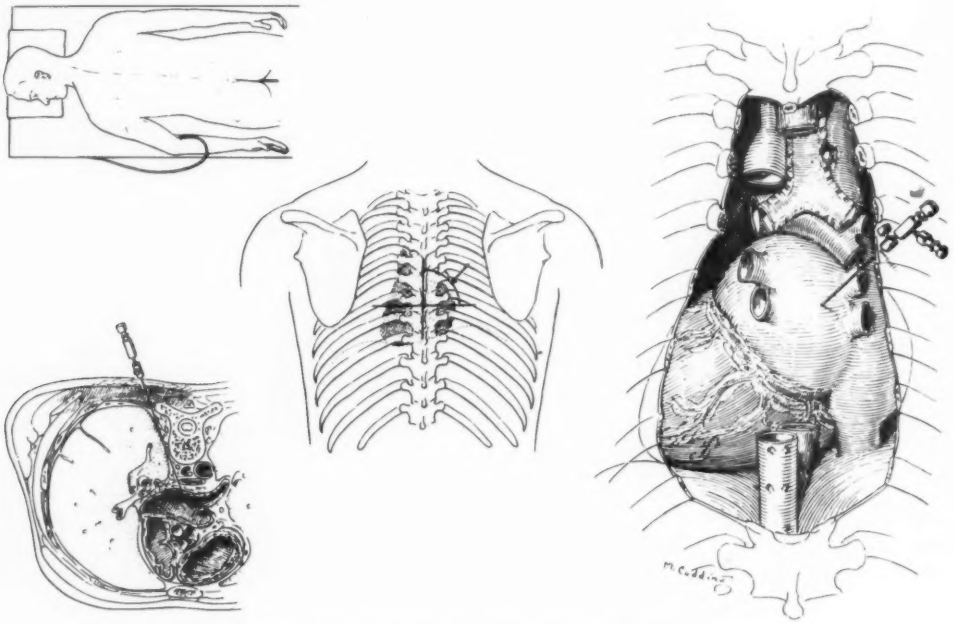

FIG. 2. Technique of percutaneous left heart catheterization.

ceal the pathologic nature of valvular disease. Furthermore, no single observer is likely to see enough fresh autopsy specimens within the limitations of memory span to allow a classification of morphologic patterns. Years ago, recognizing the limitations of the "classic method" of studying postmortem specimens, we began taking colored motion pictures of the intact valve without destroying the valve ring. These films preserve an impression of color and texture otherwise impossible. When several are viewed together a concentration of experience results which leads to a better comprehension of the basic morphologic patterns of aortic stenosis. More recently the McMillan pulsator has added kinetic to morphologic evaluation.[30]

Types of Valve Deformity: In contrast to most textbook descriptions, we have found that the most common pattern of adult calcific aortic stenosis is fusion of the anterior and posteromedial cusps with little or no adherence of the edges of the noncoronary cusp. This results in a *bicuspid valve* with only a rigid, narrow slit for an opening. Nevertheless, an area of mobility or "hinge zone" can almost always be identified near one of the commissures, often the posteromedial one. This is present in approximately 30 per cent of the cases.[13]

The other two major morphologic patterns encountered are illustrated in Figure 3. The *tricuspid form*, which has also been found in approximately 30 per cent of clinical and autopsy specimens we have examined, bears three clearly defined commissures. The third major classification is the *punctate form* which produces the most advanced narrowing with an orifice that may be no larger than a match head. Obviously there are many intermediate gradations of these basic patterns but the outstanding common denominator of all adult aortic stenosis is massive calcification and absence of mobility.

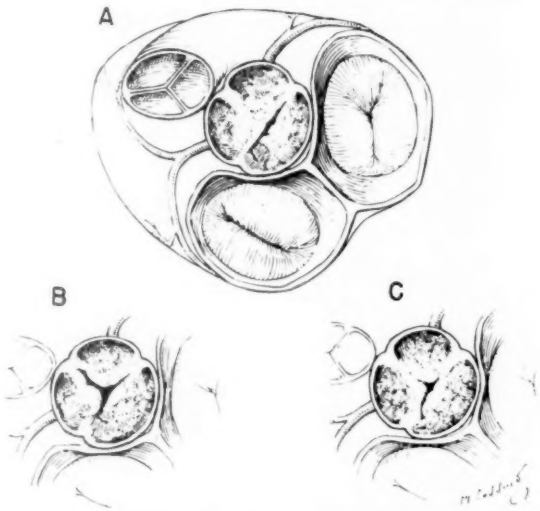

FIG. 3. Common pathologic patterns of calcific aortic stenosis. A, *bicuspid*, approximately 30 per cent of patients. B, *tricuspid*, 30 per cent; C, *punctate*, 10 per cent; remainder are combinations of these types. From: HARKEN, D. E. The surgical correction of calcific aortic stenosis in adults. *J. Thoracic Surg.*, 36: 759, 1958.

The advanced nature of these pathologic processes has made aortic valvular surgery difficult and has discouraged many. It appears that even when the commissures are effectively broken open the residual immobility of the cusp might preclude effective valvular function. This "inoperable state" seems to be relative, and defeat is less frequent as experience increases. Even so, there remain inherent limitations to correction by fracture-dilatation and mobilization that compel continuing search for suitable aortic valve prostheses.

Mechanics of Valve Closure: The mechanism of valve closure in calcific aortic stenosis is quite unlike that of the normal valve. The surgeon undertaking corrective surgery must understand both mechanisms and their combinations. After observing casts of normal aortas and aortic valves in action on the McMillan pulsator, the mechanics of normal leaflet closure have been clarified (by V. B.).[16] The cusps may be thought of as individual pouches, the free margins of which are supported by a narrow band of thickened tissue. By virtue of the fact that the point of attachment of these bands to the aortic wall is at a higher point than the center of the valve during closure, they serve to strengthen the filmy cusp tissue much as the cables of a suspension bridge. Without such support it seems inconceivable that the normal cusp would have sufficient strength to support the diastolic pressure load, even though they tend to support one another as they come into contact over a relatively broad area.* It is thus obvious that any interference with the lateral attachment of the cusps may produce regurgitation if sufficient mobility remains.

A very different valve closing mechanism may operate in tricuspid or bicuspid calcific stenosis. Here there is generally a pyramidal

* In normal operation the tissue stresses are all in tension; there are no compressive stresses, and there is no column action.

calcific mass rising to a pinnacle centrally that is higher than the point of origin of the leaflets on the aortic wall. Here closure of the valve occurs by interlocking of the rigid leaflet margins in a "keystone" fashion. The force of back pressure is then transmitted through the rigid leaflets to the aortic wall.

Frequently the two closing mechanisms are seen in different areas of the same valve. There may be some residuum of the suspension closure in one commissure and the acquired mechanism of keystone closure in another. In any event it became apparent that knowledge of these mechanisms is essential to effective correction of stenosis, without the production of insufficiency. Finally it is apparent that once leaflets are mobilized they may close more effectively than prior to surgery. Thus minor degrees of regurgitation may be improved as the dominant stenotic lesion is corrected.

Background for Current Surgical Techniques

Although sporadic attempts at aortic valvular surgery were made in the past, beginning with the dramatic report of Tuffier[31] in 1913, it was not until the past few years that any consistent attempts have been made to treat this lesion surgically. In 1950 one of us (D. E. H.) attempted high transventricular digital exploration of the aortic valve. This attempt was predicated on some knowledge of the pathologic process and previous experience with the removal of foreign bodies from the heart. The patient died from uncontrollable hemorrhage from the ventriculotomy. This emphasized the great technical problem presented by a hypertensive left ventricle in failure.

Transventricular Approach: Following this we passed through a phase of transventricular surgery inserting a larger instrument through the apex of the ventricle which dilated the valves and attempted commissural fracture by virtue of size alone. With this instrument it was possible to excise a 1 mm portion of the posterior or noncoronary leaflet in order to provide another "hinge zone" for the markedly stiffened valve. At that time we were willing to accept a limited amount of aortic insufficiency to relieve stenosis. This popular misconception was reminiscent of early mitral valve surgery. Our own and other subsequent experience has emphasized the importance of avoiding regurgitation which is just as poorly tolerated in the aortic valve as in the mitral valve. In addition to these considerations, ventricular cardiotomy is poorly tolerated by the patient with aortic stenosis because of myocardial irritability which may lead to ventricular fibrillation. Another difficulty in the transventricular approach is that of hemostasis in a muscle that holds sutures poorly. Even though these technical problems can now be reduced greatly by improved smaller valvulotomes and better suture placement, one still is left with a method that is poorly controlled. One cannot really tell where he is placing the fracture and how much leaflet mobility is being affected. In the final analysis this is a cardinal limitation.

Of 23 patients with pure aortic stenosis operated on by the original transventricular route, 10 (or 43 per cent) died from the operation and 10 more have subsequently succumbed. Two of the survivors were reoperated upon via the transaortic route but only one survived.

Recently with improved valvulotomes we have returned to the transventricular approach in 11 patients who had combined aortic and mitral stenosis. Again, although the technical aspects of the ventriculotomy and valvulotome manipulation have become exceedingly simple, the correction of the aortic stenosis has not been as satisfactory. At times the stenosis has not been relieved or significant regurgitation has been produced. The instruments and ventriculotomy are not the problem, but the lack of control is a fundamental defect.

Transaortic Approach: Following the demonstration by Swan[11] that the aortic valve could be successfully approached from above through an operating tunnel sutured to the wall of the aorta, we have employed a modification of this technique in 100 cases. Tunnels made of cotton, nylon, and polyethylene-lined nylon have been used. It was not until Hugh Wilson,* while working with us, suggested the use of an Ivalon sponge tunnel that a consistently satisfactory hemostatic method was developed. This tunnel will be described in the section on technique. It is important to emphasize that the development of this specific tunnel has marked the beginning of a more consistently satisfactory experience with the transaortic procedure. The hurried surgery, dictated by inadequate hemostasis, undoubtedly accounted

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in part for the higher mortality and poorer rehabilitation in the earlier operations of this series. The limitations of other materials such as pericardium, rubber, blood vessels and cloth, become more conspicuous when one has worked with the Ivalon tunnel. An unhurried and deliberate valve manipulation may be carried on for as long as 45 minutes if necessary. This is a gratifying contrast to the open techniques or other operative tunnels. Furthermore, open techniques which carry the additional risk of the bypass itself, especially when the left ventricle is in failure, must be regarded as only relatively direct vision procedures. It is difficult enough to bring the aortic valve into view, but when it is irregular and calcific, the location of the stoma may be elusive. This is not the case when the closed technique is used. Here the jet of systole can be the guide to the orifice. Finally, the degree of mobilization is better controlled when the leaflets are activated by systolic ejection. This can be assessed by the intra-aortic finger. The tendency to overcorrect at the expense of regurgitation is a menace in either operation but is much better controlled by palpation than in the passive state of open direct vision surgery.

Experience in the autopsy room as well as at the operating table leads to the conclusion that the most effective maneuvers are not those made in the flaccid heart under vision but those possible in the beating heart with the palpating finger as the guide. The cardinal maneuver is the insinuation of the finger under the calcific leaflets with pressure directed upward in the line of a fused commissure from the ventricular side. The undersurface is uniformly smoother and the commissural creases are better identified in this way. Such a mobilization is less likely to disrupt the commissural moorings to produce regurgitation or to release calcific emboli.

Technique

With the patient in the supine position a vertical midline incision is made from approximately the level of the first intercostal space down to and beyond the xiphoid. At the upper end a curving horizontal limb is made so that a flap of skin and platysma can be elevated above the clavicles. The sternum is then split slightly to the left of the midline so that when the retractor is inserted the weaker left side spreads to a greater extent, thereby exposing the left ventricle (Fig. 4).

Neither pleural space is opened, with the result that

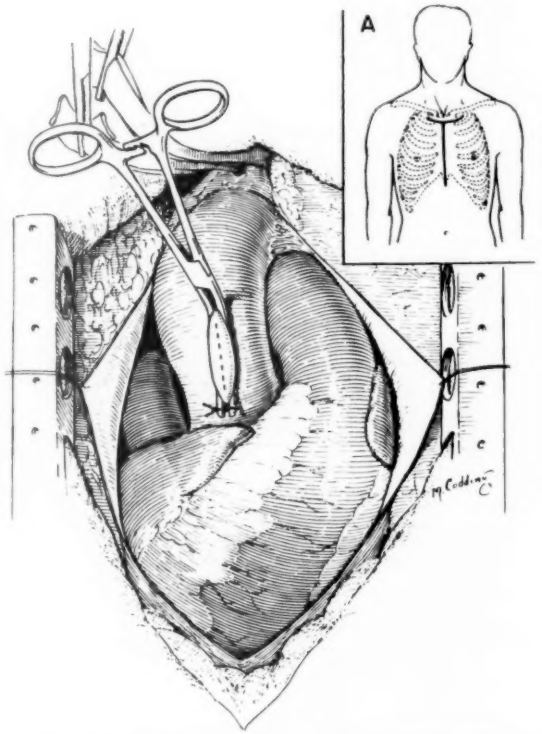

Fig. 4. Incision and exposure for transaortic valvuloplasty. Insert: Midline sternotomy. Curved Atraugrip clamp excludes a segment of ascending aorta. Rubber dam drains about right innominate and left common carotid arteries.

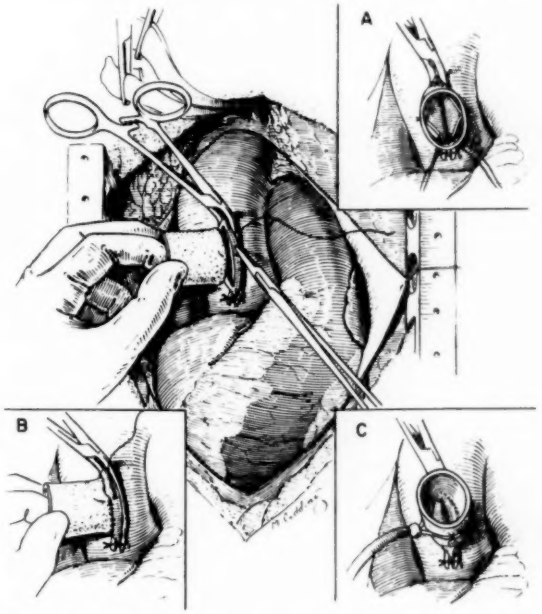

Fig. 5. Ivalon operating tunnel is sutured to exclude portion of aorta with interrupted, then continuous silk sutures.

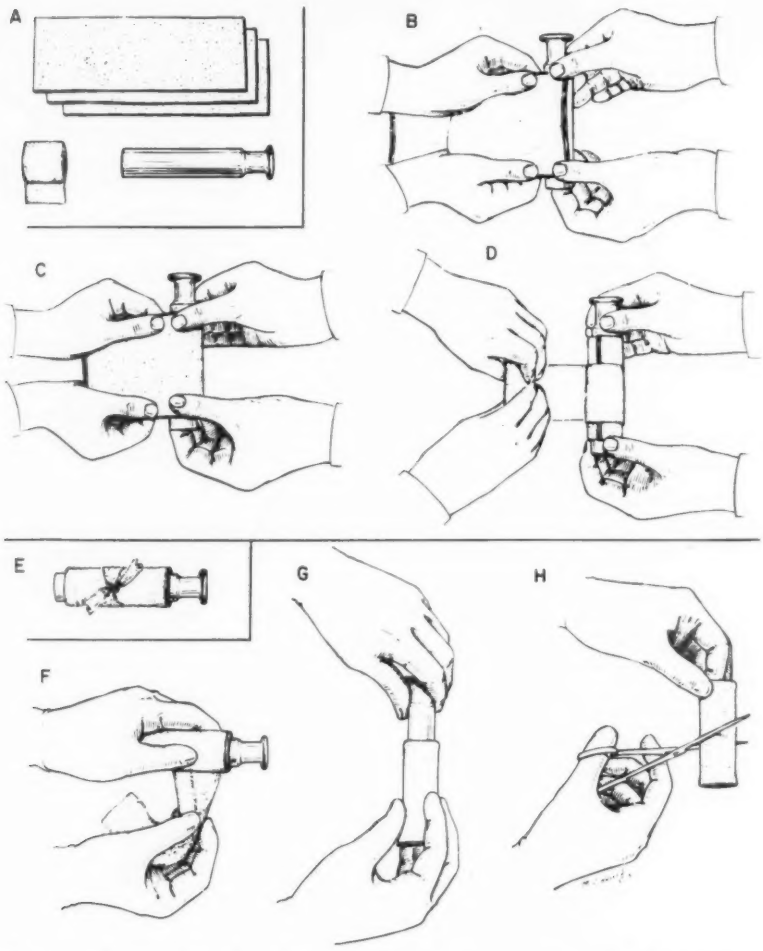

FIG. 6. Preparation of Ivalon operating tunnel: Three strips of moistened Ivalon are firmly wrapped about the plunger of a 30 cc syringe. The strips are compressed with a bandage. The tunnel is boiled for 30 minutes to mould. The tunnel is trimmed as indicated at operation.

adequate ventilation and oxygenation are much more readily accomplished. Our anesthetists have reported that the management of these patients has been much simpler with this incision than with the posterolateral thoracotomy formerly used for the transventricular approach.

Once the sternum is spread, excellent exposure of the anterior surface of the heart, the arch of the aorta and the great vessels is obtained. The pericardium is opened longitudinally up to the point of insertion on the arch of the aorta. Pericardial marsupializing sutures are used to support the heart in the field. The right innominate and left common carotid arteries are isolated and rubber dam drains placed about them (see Fig. 4). During the valvular manipulation, if calcium is broken into or released, the circulation to the brain is interrupted by traction on the rubber drains. The occlusion is limited to 15 to 20 seconds with rest periods of at least two minutes. Our first patient operated on by this approach without isolation of the head vessels died of massive calcium embolization, but the use of cerebral circulatory occlusion during manipulation since that operation has prevented any further serious cerebral embolic complication.

Aortotomy: A curved Atraugrip* clamp is applied to the anterior surface of the aorta as close to the base of the heart as possible without compromising the right coronary artery (Fig. 5). A #5 braided silk tie is applied to the notched tips of this clamp to assure uniform coaptation of the blades. An incision that is slightly larger than the index finger is then made in the isolated segment of the aorta. Incision on the anterior surface of the aorta rather than on its lateral aspect is preferred so that the aortotomy

* George P. Pilling & Son Co., Philadelphia, Pennsylvania.

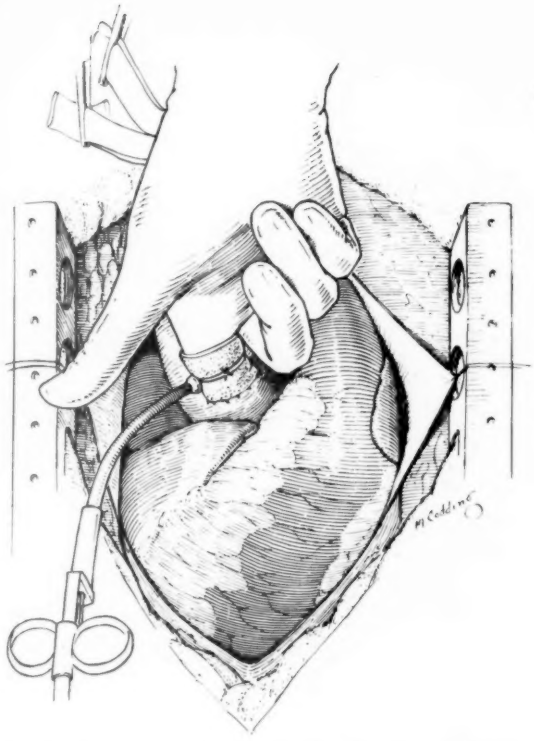

FIG. 7. Ivalon tunnel in position sutured to aortotomy. Operator's finger in aorta above valve.

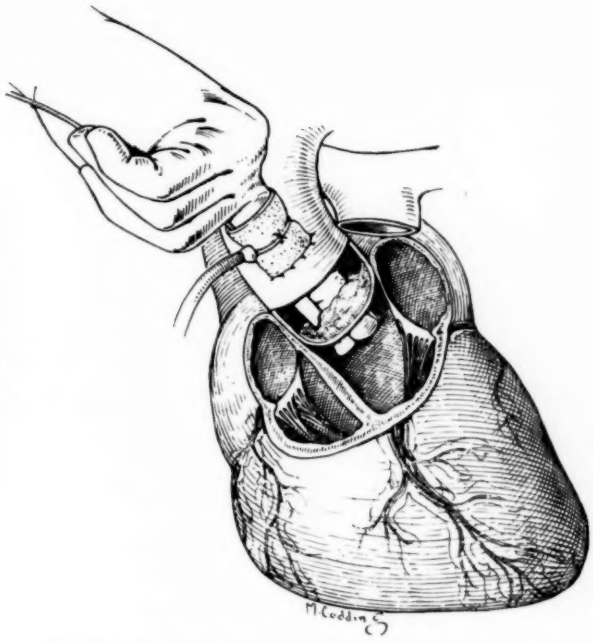

FIG. 8. Number 10 valvulotome in place.

is supported by the sternum postoperatively. No aneurysm at the aortotomy site has developed with this incision although one late death occurred from an infected aneurysm when a right thoracotomy approach had been used.

Ivalon Tunnel: The Ivalon tunnel employed is prepared by wrapping three or four layers of moistened, thin-sliced Ivalon on the barrel of a 30 cc syringe (Fig. 6). The bottom edge of this tunnel is built up to a slightly greater thickness than the remainder for the purpose of holding the sutures. This is tightly wrapped with gauze to compress it. Sterilization is accomplished by boiling for 30 minutes. The individual operating tunnel, 3 or 4 cm in length, is cut from this tube and sewn to the lips of the aortic incision with #000 braided silk, interrupted sutures through the full thickness of the aorta. These are first placed at the four quadrants of the incision and the natural elasticity of the tunnel holds the aortic edges open for easy and rapid suturing. The second layer consists of a row of continuous silk suture which forms a water tight seal between the external edge of the Ivalon tunnel and adventitia of the aorta (Fig. 5). Although some oozing through the tunnel may occur when the clamp is first released, the interstices quickly fill with clots and no further bleeding occurs. With proper application of the tunnel a bloodless manipulation of the valve can be carried out except for the small leak on first insertion of the finger and on its removal from the tunnel. Oozing about the finger is controlled by umbilical tape on the outside of the tunnel and held in a Rumel tourniquet (Fig. 5C).

Mobilization of Valve Leaflets: Following insertion of the finger, the orifice of the aortic valve is sought by following the systolic jet (Fig. 7). It was originally believed that the bicuspid valves consisting of two inflexible ledges could be mobilized best by separating the lateral attachments of the aortic wall to effect a "trap door." It soon became apparent, however, that such mobilization could result in promptly fatal insufficiency. This accounted for three early deaths on the operating table.

Recognizing the mechanisms of closure, every effort is made to open the existing commissures accurately. After the finger has been inserted through the valve, pressure is made on the commissures by lifting from below upwards. In this way separation is possible in the majority of instances. The mobilization may be improved by the use of the #10 valvulotome or by the use of a flat two or three-bladed dilator which is slipped into the aorta beneath the operator's glove (the tip of the index finger of the glove having previously been cut off) (Figs. 8 and 9).

Following mobilization of the leaflets the shock of valve closure can often be felt on the external surface of the aorta and in some instances coexisting aortic insufficiency has been found to be diminished as estimated grossly by the degree of reflux and collapse of the aorta in diastole. The entire effort is directed toward mobilization of the heavily calcified cusps

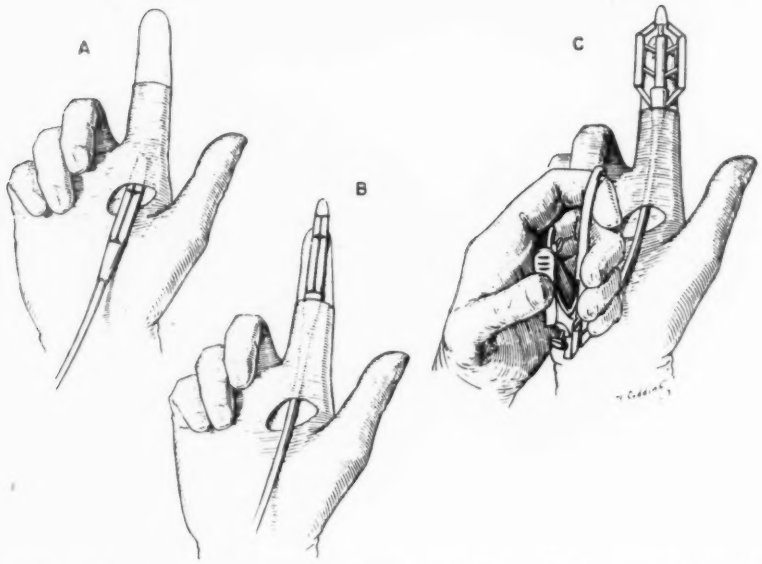

FIG. 9. A bicornuate or tricornuate dilator may be inserted through glove and operating tunnel.

to allow a keystone type of closure which will prevent regurgitation.

Following manipulation the operator's finger is slowly removed while an assistant replaces the curved Atraugrip® clamp. If this is not placed sufficiently deep on the aorta, the Satinsky model of the Atraugrip clamp can be placed just proximal to it. Whichever clamp is used, a #5 silk suture is again used to stabilize the blade ends.

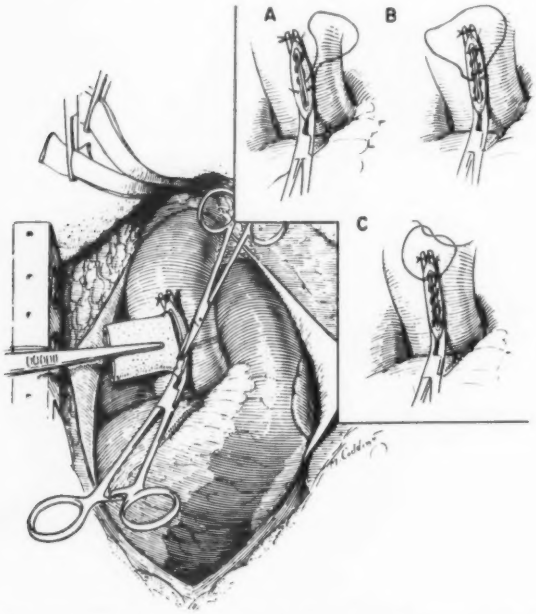

FIG. 10. The curved Atraugrip clamp has been reapplied and tip secured with a #5 silk suture. The tunnel is removed. The aortotomy site is closed with two layers of silk suture.

Closure: The tunnel is then removed and the aortotomy closed with a continuous everting mattress suture of #00000 silk reinforced with an over and over stitch of the same material (Fig. 10). A Gelfoam® sponge is placed over the aortotomy and the pericardium closed.

Closure of the sternotomy is effected by four or more pairs of crossed 30 gauge stainless steel wires through or around the sternum. Greater stability is assured by crossing pairs of wires rather than using them horizontally. Any tendency to vertical slipping of the two edges of the divided sternum is thus corrected by the obliquely placed wires which force the edges of the sternum more tightly together.

Hemostasis: The sternotomy incision unquestionably bleeds more than other thoracotomy incisions. Careful hemostasis particularly on the internal surface of the sternum and along its edges with cautery, bone wax, and transfixion sutures will minimize this problem. In addition Gelfoam sheets are liberally used between the sternal edges. Originally we used a drain at the inferior portion of the wound, but in order to avoid retrograde infection an opening is now routinely made into the right pleural space with a counter incision in a dependent portion of the pericardium and a water-sealed posterolateral drainage tube in the pleural space.

Postoperative Management: Postoperatively these patients are maintained on oxygen for two or three days and not allowed out of bed until their fourth day. They are ambulated on the seventh day. Blood loss in these procedures averages 1,500 cc by sponge weight and measurement of blood aspirated

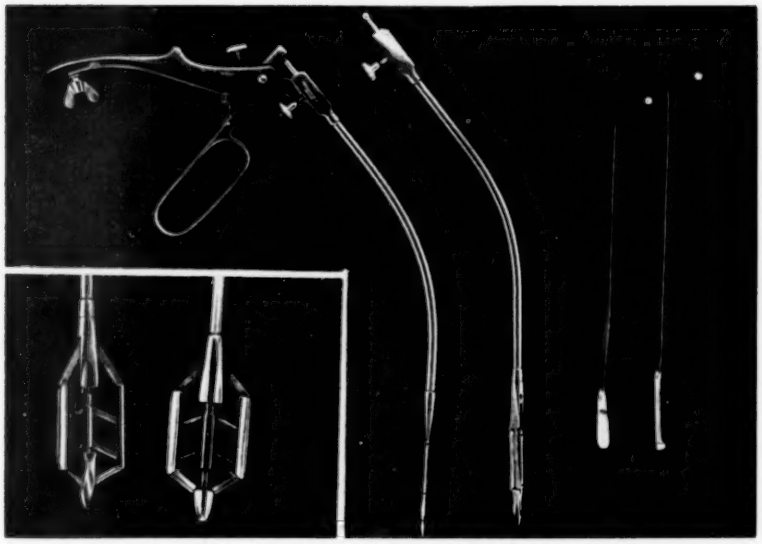

FIG. 11. Various valvulotomes are used through the glove and operating tunnel. Single and double blade dilator, smooth dilator and sawtooth valvulotome (Codman and Shurtleff, Inc., 104 Brookline Avenue, Boston, Massachusetts).

by suction. In order to compensate for the blood that remains in the pleural space, mediastinum, or on the drapes, 1,000 to 1,500 cc more than the measured loss is given. In the postoperative period drainage through the thoracotomy tube is replaced volume for volume with additional blood. The natural tendency is to err on the side of inadequate replacement in these patients, but if increased venous pressure is not produced (rare in our experience), it is better to assure adequate blood volume and oxygen-carrying capacity.

RESULTS

This report covers our first 100 consecutive transaortic operations for *calcific aortic stenosis in adults*. While the usual findings of syncope, pain, and auricular fibrillation were commonly encountered, the principal and most urgent indication for surgical intervention was left ventricular failure. The evidence for this ominous circulatory development varied from progressive dyspnea on exertion in spite of good medical treatment to overt congestive failure with refractory fluid retention. One or the other of these findings was present in 88 per cent of the operative series.

In the surgical series there were 16 *operative deaths* and 12 *late deaths*, indicating that even with surgery all patients do not do well. Nevertheless, the mortality from operation has steadily declined so that there have been only 5 deaths in the last 60 cases. Of the 50 survivers who have been followed up by Dr. Laurence B. Ellis for more than six months (up to 36 months), 86 per cent are improved. The restoration by operation of the majority of these patients to a comfortable existence is a sharp contrast to the outcome in the control series. As previously mentioned, in a group of 54 patients in which operation was recommended but not carried out for various reasons, 49 were dead within six months.

The detailed clinical and hemodynamic evaluation of surgical results is the subject of another communication.[14] It is sufficient to point out here that not only can the majority of survivors be improved clinically, but objective evidence of hemodynamic improvement is also present.

SUMMARY

(1) The life cycle of adults with calcific aortic stenosis is discussed. The ominous significance of left ventricular failure, even as manifested by dyspnea on exertion, is emphasized.

(2) A transaortic technique for the correction of this condition is presented. The substantial advantages of using a special Ivalon operating tunnel are reviewed.

(3) In the first 100 cases there have been 12 late deaths and 16 operative deaths. The operative mortality has been reduced to 5 in the last 60 consecutive patients.

(4) Eighty-six per cent of the surviving

patients followed from 6 to 36 months are improved.

(5) The gratifying salvage from this fatal disease encourages further use of this method of surgical relief.

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The Accomplishments of Open Heart Surgery

The Status of 35 Patients 18 to 36 Months Following the Operation*

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THE USE OF extracorporeal circulation is now assured a permanent role in the treatment of certain cardiac lesions. Due to the relatively recent advent of this type surgery, there have been few reports except of the immediate results. Accordingly, a review of our first 35 surviving patients, operated on with the aid of total cardiopulmonary bypass 18 to 36 months ago, was undertaken.†

The first successful, complete bypass of the heart and lungs was accomplished in 1953 by Gibbon who was able to repair an atrial septal defect in a young woman, using an open technique and a filming type oxygenator. Lillehei, in 1954, following experimental work in animals, utilized the method of cross circulation to repair a ventricular septal defect, using an open technique and cross transfusion with a human donor. This work was followed by the development of a bubble type oxygenator by DeWall[1] and others.[2,3] Preliminary to these complete bypass operations, pumps had been developed and successfully used for bypass of each side of the heart alone by Dodrill,[4] Bailey,[5] and others.

SELECTION OF PATIENTS

Of these 35 patients, 23 were suffering from aortic stenosis and 2 from aortic insufficiency. Of the ten patients with congenital defects, three had congenital aortic stenosis. There were four patients with the tetralogy of Fallot and three with ventricular septal defects.

The technique employed in all these cases involved the use of the Friedland-Gemeinhardt oxygenator, a modification of the DeWall apparatus (Fig. 1). Two Sigmamotor pumps were used to replace, respectively, the right and left ventricles. The bodily venous return was withdrawn from the venae cavae through separate catheters, and the oxygenated blood was returned to the arterial circuit by way of a subclavian catheter. An additional Sigmamotor pump was used in some of the patients for retrograde perfusion of the coronary sinus with oxygenated blood.[6] Throughout the entire series, the rate of extracorporeal flow was maintained at nearly the patient's normal cardiac output (adult average over 3,000 cc per minute). The last patient in the series, operated on August 21, 1957, was perfused by a new principle involving the use of the patient's own lungs as an oxygenator.[7] The technique of cannulization in this case was as shown in Figure 2.

All of these patients were in a supine position when explored using a transverse incision through the bed of the third or fourth rib, and transecting the sternum at that level. Elective cardiac arrest was not used in this series. Since then arrest was used for a period of one year and now has been abandoned.

ACQUIRED AORTIC STENOSIS

In this group there were 21 men and 2 women. The 2 women were aged 42 and 53 years, respectively. The ages of the men ranged from

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† At the time these patients were operated on our mortality with open heart surgery had been 42 per cent in this relatively small beginning series. The current mortality with open heart surgery at this clinic is 18.2 per cent. The total series now is in excess of 400.

TABLE I

The Age Distribution of 23 Patients with Acquired Aortic (Calcific) Stenosis Operated on by an Open Technique

Age (yr)	Patients (no)
10–19	0
20–29	1
30–39	6
40–49	9
50–59	7
TOTAL	23

29 to 58 years, and they were grouped by decades, as shown in Table I. All the patients reported in this study exhibited manifestations of severe and progressive aortic stenosis. The predominant clinical symptoms were those of shortness of breath, pain, tightness in the chest, and syncope. All the patients with acquired heart disease were classified by our cardiologists into the American Heart Association functional class II or III, being about equally divided between those two groups.

All the patients in this series had preoperative measurement of the pressure gradient (systolic) across the aortic valve to confirm the necessity for surgery. In most cases this was accomplished by the technique of left heart catheterization via the posterior transthoracic puncture route,[8] but in four cases the brachial arterial pressure was measured by puncture and the

TABLE II

Complete Pre- and Postoperative Physiologic Data* in a 45-Year-Old Man (E.S.) with Severe Calcific Aortic Stenosis

	Gradient (mm Hg) (by planimetry)	Flow (cc/sec)	Systolic ejection time/beat (sec)	Calculated functional valve area (cm^2)
Preoperative	118	167	0.28	0.34
Postoperative	32	192	0.20	0.8

NOTE: This patient was operated on in our early series before the present technique of subtotal excision of calcium from the cusp leaflets was developed. He really represents the effect only of accurate commissurotomy upon the circulatory dynamics. We believe that the results of commissurotomy plus valve sculpturing in terms of final valve area will be much better (see Figure 3).

* The data in this and the subsequent tables were obtained in the Brith Sholom Cardiopulmonary Laboratories of Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania.

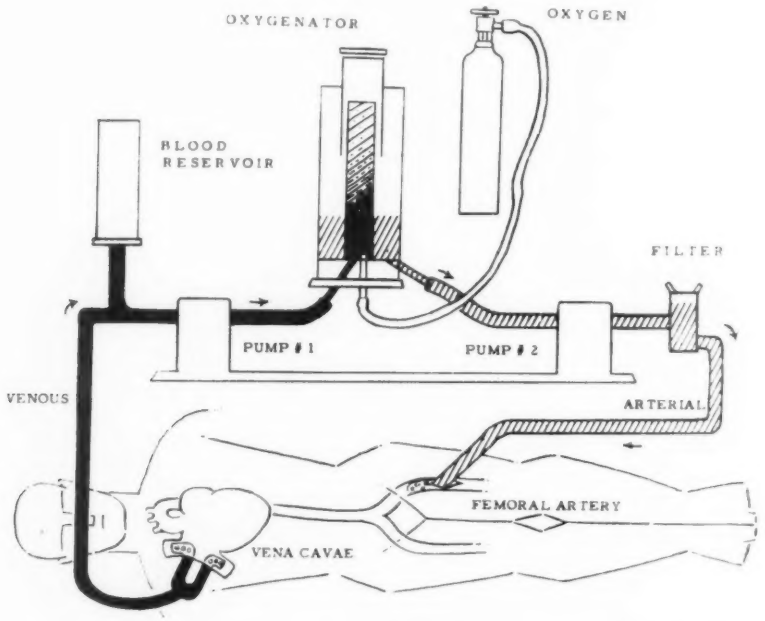


FIG. 1. The "standard" cardiopulmonary bypass. Unoxygenated blood from the venae cavae (shown in black) is pumped through a bubble oxygenator and returned via a second pump into a major artery (the subclavian has now been supplanted by the femoral for this purpose).

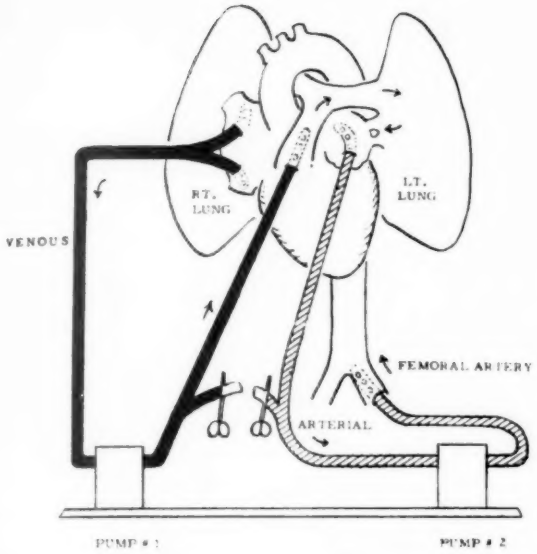


FIG. 2. The "autogenous lung" cardiac bypass. In this method the patients' own lungs are used to oxygenate the blood. This particular technique, developed by Dr. Gumersindo Blanco of the Bailey Thoracic Clinic, avoids the trauma to the blood elements caused by mechanical oxygenators, and allows successful complete cardiac bypass for periods as long as two hours. Venous blood (in black) is collected by gravity drainage from a single large or double catheter in the right atrium, and passed into the pulmonary artery. Oxygenated blood (shaded lines) is similarly drawn from the left atrium and passed into the femoral artery. An oxygenator (connected to ends of clamped tubes) is included in the circuit for emergency use if indicated.

ventricular pressure was obtained by direct anterior subxiphoid puncture of the left ventricle because it had been elected to perform simultaneous ventriculographic evaluation of possible mitral regurgitation. The measurement of the gradient across the aortic valve is understood to be only one part of the physiologically threefold determination of the degree of obstruction at the valve. The measurement of flow and of the period the valve was open during each cardiac cycle was not always performed but such complete evaluation is illustrated in Table II. This shows the complete pre- and postoperative physiologic data as determined by combined right and left heart catheterization of a 45-year-old man who had severe calcific aortic stenosis. With the reservation that the measured gradient only approximately indicates the degree of physiologic obstruction, the results of pre- and postoperative physiologic studies in these patients are presented in Table III.

TABLE III

Catheterization Results in Congenital and Acquired Calcific Aortic Stenosis in Those Patients Studied Pre- and Postoperatively

Case No.	Age (yr)	Type of lesion	Date of operation	Aortic valve systolic gradient —(mm Hg)— Preoperative	Postoperative
1, B.G.	14	C	4/12/56	57	22
2, C.M.	57	A	5/29/56	54	17
3, J.C.	14	C	6/21/56	145	43
4, F.S.	45	A	7/13/56	200	70
5, J.H.	38	A	7/18/56	152	70
6, F.A.	44	A	7/25/56	146	8
7, J.J.	53	A	11/2/56	80[a]	39[a]
8, G.W.	37	A	6/12/57	85	40
9, G.F.	42	A	8/9/57	78[b]	72[b]
10, E.S.	45	A	8/21/57	118	42

NOTE: C = congenital. A = acquired.

[a] By planimetry (all other gradients measured "peak to peak") and ventricle to brachial artery.

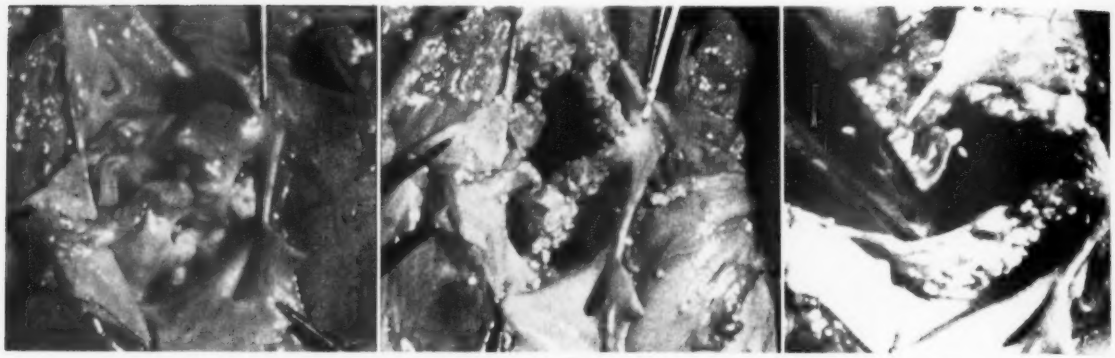
[b] Increase in flow 231 cc to 292 cc per systolic ejection period second, in less time (0.34 second to 0.20 second per systole). This patient has had a remarkably good result clinically. He is asymptomatic and working full time.

COMPLICATIONS OF SURGERY

One patient had continued bloody pleural drainage postoperatively which required reexploration 24 hours after surgery, at which time the hemorrhage was observed to come from the aortic suture line. This was repaired, the thorax was reclosed and no further difficulty was encountered. In one patient with congenital aortic stenosis, a significant amount of aortic insufficiency was produced so there was a progressive postoperative drop in the diastolic blood pressure to a level of 10 mm Hg following surgery.

Local wound complications occurred in five patients. In two of these, small draining sinuses developed which healed after a short time. Two more had hematomas of the wound which became infected and resulted in nonunion of the sternum. In an additional patient, nonunion of the sternum occurred without infection. Two of these patients with nonunion are still complaining of discomfort.

A number of the patients had pulmonary complications. Tracheotomy was necessary in two patients of the aortic series. In many of them, excessive tracheobronchial secretions necessitated bronchoscopy in addition to multiple tracheal aspirations.



A B C

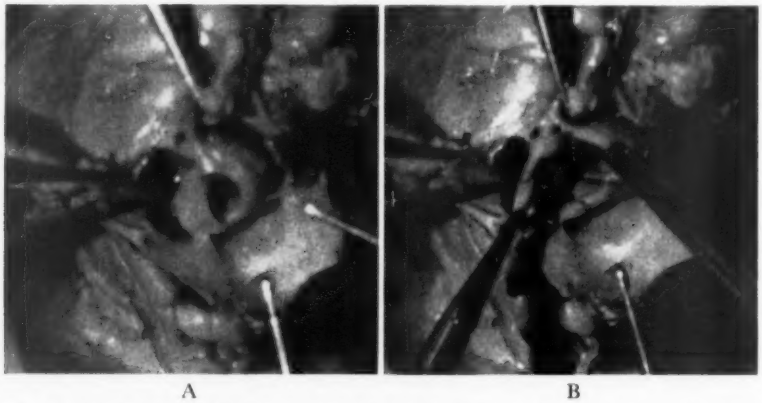
FIG. 3. A, a typical aortic valve with severe calcification completely immobilizing the cusps, and obliterating the commissure to the right. B and C, the same valve similarly oriented after removal of the calcium from the concave side of each cusp, and incision of the fused commissure. Two leaflets are now freely mobile.

In several of the patients, manifestations of cerebral disturbances developed postoperatively. One patient (T. H.) had evidence of air embolism to the middle cerebral artery as determined by neurologic examination. Recovery was complete. Another became frankly psychotic and agitated, but he improved before discharge. A third was confused for three days following surgery and had a minor facial weakness. It was thought that this latter episode might have been related to embolism occurring at the time of surgery. In one case, there was difficulty with the circulation in the left arm, presumably due to acute arterial insufficiency from the left subclavian cannulation. This subsided with physiotherapy.

Hemolytic jaundice developed in one patient during his hospital stay. Hepatitis occurred in another patient four months after surgery, requiring rehospitalization. Both patients recovered.

POSTOPERATIVE PHYSIOLOGIC STUDIES

Although subjectively the patients with aortic stenosis were completely relieved, or at least greatly improved symptomatically, the catheterization data in the patients who have returned for study show that in these early open procedures for aortic stenosis we failed to bring about a return to normal pressure and flow relationships (Tables II and III). Because of the severe degree of valvular calcification, fibrosis and rigidity of the valve leaflets, a complete obliteration of the pressure gradient was



A B

FIG. 4. A, the typical appearance of the pulmonary valve in pure pulmonary stenosis in a patient with tetralogy of Fallot. Although the valve at first appears to be a simple cone, in fact, each rudimentary commissure can be recognized by a tissue bridge raphe extending to the arterial wall. B, the same valve similarly oriented after division of the commissures. A good opening may be obtained quickly and easily without production of insufficiency. Infundibular stenosis is, of course, more common than such valvular obstruction in the tetralogy of Fallot.

TABLE IV

Catheterization Data in a Patient with Tetralogy of Fallot (J.J.), Age 3 Years, Operated on March 28, 1957

	Immediately preoperative	6 months postoperative
Pressures (mm Hg)		
Main pulmonary artery	6/3	18/8
Right ventricle	100/0	26/4
Brachial artery	106/70	87/55
Right atrium	(0)	(4)
Pulmonary venous capillary	(2)	—
Oxygen contents (vol %)		
Superior vena cava	11.8	10.6
Inferior vena cava	9.2	—
Right atrium (high)	11.6	11.6
Right atrium (middle)	12.1	—
Right atrium (low)	11.6	11.6
Right ventricle, tricuspid	8.2	11.5
Right ventricle, apex	11.1	10.3
Main pulmonary artery	8.8	11.1
Right pulmonary artery	—	11.1
Brachial artery capacity	19.4	16.3
Brachial artery saturation (%)	73.3	91.4

not obtained. (In our more recent experience with such valves, to be reported later, a new technique has been developed for removing the calcium and much of the scar tissue from the concave side of each cusp. The "sculpturing" restores mobility and function to the valve to a near-normal extent (Fig. 3).)

Tetralogy of Fallot

The ages of the four patients with tetralogy of Fallot were 2, 3, 15, and 22 years, respectively. Three of the four had infundibular stenosis; one had a valvular stenosis; none had combined stenosis. All the defects were in the membranous portion of the interventricular septum and averaged approximately 1.5 cm in diameter. In each case, the ventricular septum was repaired with simple or mattress sutures. In one case this was supplemented by Ivalon pledgets to reinforce the mattress sutures. In no case was a conduction bundle disturbance initiated by the repair. The protruding portion of the supraventricular wall was resected in the three cases in which obstruction was of the infundibular type. In the patient with purely valvular stenosis, the pulmonary artery was opened and the commissures were incised along the line of congenital fusion (Fig. 4). All four patients had been catheterized preoperatively and in each case the right ventricular systolic pressure was found to be over 75 mm Hg.

In the two patients in whom postoperative catheterization studies have been carried out, complete cure has been demonstrated by the physiologic data (Tables IV and V).

Ventricular Septal Defect

Our first attempt at closure of a ventricular septal defect was made in 1952.[9] This patient was treated without the use of an open technique, a pedicled pericardial plug being pulled through the ventricular defect to block it. Because of significant obstruction to the right ventricular outflow tract, it later was necessary to remove the tampon in the defect. Our first successful modern closure of a ventricular defect took place in July 1956, in an 18-year-old boy. Two additional patients are included in our 18- to 36-month follow-up. In each of these cases, the defect as shown in Table VI has been approximately 1 to 1.5 cm in diameter and was closed with simple sutures. The demonstration by catheterization of complete cor-

TABLE V

Surgical Findings and Results in Four Patients with the Tetralogy of Fallot (Postoperative)

Patient, Age (yr), and Sex	Date	Defect (cm)	Type of pulmonary obstruction	Closure	Present condition
J.J.,[a] 3, F	3/28/57	1.5	Infundibular	Simple suture	Excellent (see Table IV)
J.E.,[a] 2, M	5/31/57	1.5 × 1.0; membranous septum	Infundibular	Mattress suture	Excellent (catheterization 5 months postop. shows cure)
S.F., 15, F	8/6/57	1.5; high membranous	Valvular	Simple	Excellent
H.V., 22, F	8/16/57	1.5; high	Infundibular	Mattress over Ivalon pledgets	Excellent (asymptomatic, working)

[a] Complete relief of both the pulmonary stenosis and the ventricular septal defect has been proved by catheterization (see Table IV).

TABLE VI

Operative Findings and Results in Three Patients with Ventricular Septal Defect Now 18 to 36 Months Postoperative

Patient and Age	Operation date	Size and location of defect	Method of closure	Complications	Result
T.H., 18 yr	7/12/56	1.5 cm; posterior, high	Simple mattress sutures	Cerebral air embolism—no permanent damage	Excellent (working)
S.S., 6 yr	6/27/57	1.0 cm; superior	Simple sutures	None	Good (normal weight gain)
C.J.,[a] 13 mo	4/2/57	1.5 cm; beneath septal leaflet of tricuspid valve	Simple sutures	None	Excellent

[a] Complete closure of the defect has been proved by catheterization (see Table VII).

rection of one such defect is shown in Table VII.

Pure Aortic Insufficiency

We now have two patients who survived more than 18 months following plication and resection of the noncoronary bearing cusp of the aortic valve. One of these patients was 37 years old, and the other 36. Both had had severe clinical symptoms, for seven years in one case and two years in the other. These consisted of shortness of breath, precordial (anginoid) pain, swelling of the feet and palpitation. Both had been on digitalis for more than one year and had received mercurial diuretics, and both were on a salt-free diet prior to surgery. The man's blood pressure was 165/40/0 mm Hg, and the woman's 150/20/0.

TABLE VII

Pre- and Postoperative Catheterization Data in a 13-Year Old Boy (C.J.) with a Ventricular Septal Defect Operated on April 2, 1957

	Preoperative	5 Months Postoperative
Pressures (mm Hg)		
Right atrium	(6)[a]	(5)
Right ventricle	59/4	36/7
Pulmonary artery	40/19	26/10
Pulmonary venous capillary	(11)	(6)
Brachial artery	124/70	136/68
Oxygen contents (vol %)		
Superior vena cava	12.3	12.6
Inferior vena cava	13.1	—
Right atrium (high)	12.6	14.6
Right atrium (middle)	13.0	—
Right atrium (low)	12.4	13.7
Right ventricle (apex)	14.1	14.1
Right ventricle (outflow)	15.8	14.1
Main pulmonary artery	15.8	13.6
Right pulmonary artery	15.8	14.7
Left pulmonary artery	15.9	14.3
Brachial artery	17.6	19.3
Brachial artery capacity	18.8	19.3
Brachial artery saturation (%)	93.6	94.8
Blood flow (L/min)	3.3	5.6
Shunt (right-to-left)	0	0
Shunt (left-to-right)	6.1	0

[a] Figures in parentheses mean average pressure.

At surgery, in both cases, the ascending aorta was opened and under direct vision, mattress sutures were placed to obliterate the region of the noncoronary bearing cusp and one-third of the circumference of the aorta. This produced a competent biscuspid valve in each case (Fig. 5). Both of these patients remain improved at the present time. Clinically and hemodynamically the evidences of aortic regurgitation are much reduced. The signs of congestive heart failure, angina pectoris, and the palpitation previously present in each case have disappeared. Aortographic evidence of complete correction in such cases is now available (Fig. 6).

Comments

The late results of corrective surgery in these early cases clearly demonstrate that certain defects which cannot be treated adequately by the techniques of closed heart surgery, now may be relieved or even "cured" with an open technique made possible by the use of the pump oxygenator. The clinical improvement in these patients has been documented by the postoperative studies which, at least in most cases, show complete cure.

It is anticipated that the results of more recent work with the open heart procedure will

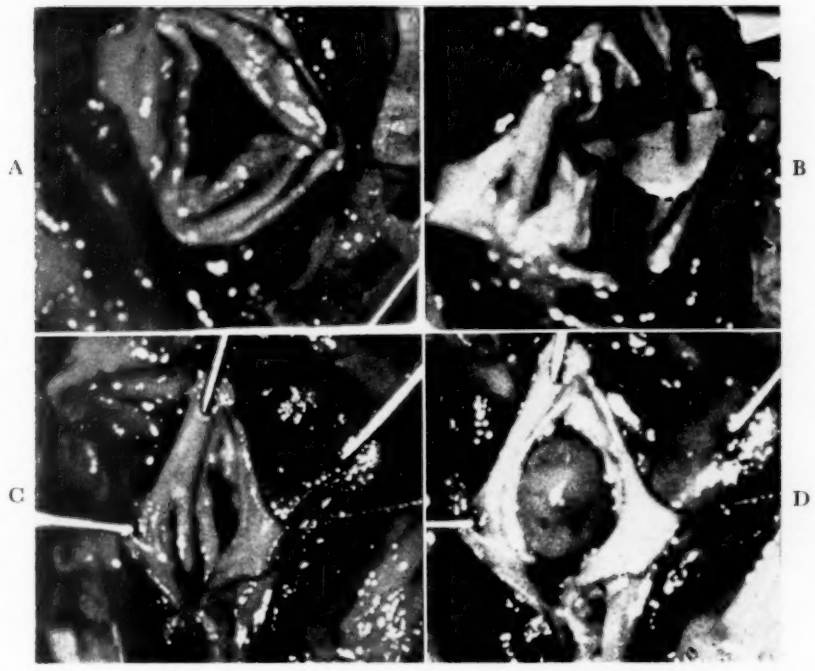


FIG. 5. A, the aortic valve in a patient with severe aortic insufficiency. The leaflet edges are rolled and shortened so that the cusps do not meet and tend to prolapse into the ventricle during diastole. B, the same valve illustrating excision of the noncoronary bearing cusp (arrow and dotted outline indicate the excised cusp). C, the valve after suture of the remaining cusps together to form a competent bicuspid valve. D, a finger is introduced through the valve from below to show the size of the aperture so produced.

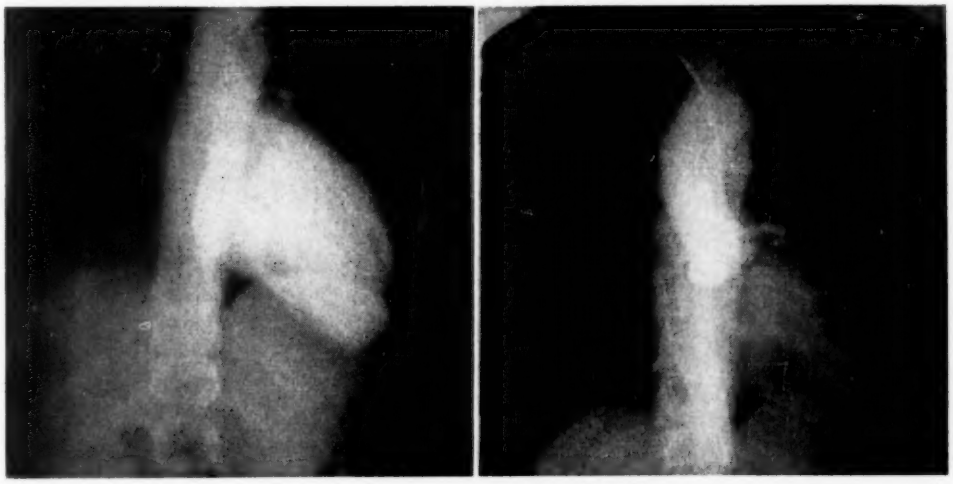


FIG. 6. Left, aortogram performed prior to surgery on June 3, 1958 on a woman with severe aortic valvular insufficiency (E.A.). The radiopaque dye injected into the aorta through a needle inserted through the suprasternal notch (and visible at the top) refluxes massively into the large left ventricle. Right, catheter aortography on the same patient performed postoperatively. At the same time interval after injection of the dye (film 6), there is beginning filling of the descending thoracic aorta. There is no reflux into the ventricle. This patient, although operated on more recently, has so far had the clinical improvement manifested by the earlier patients.

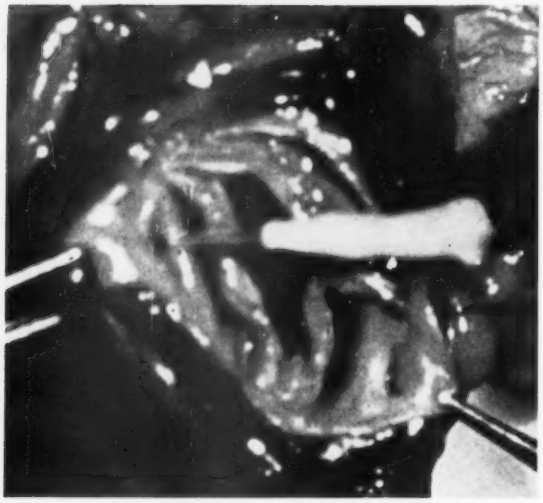


FIG. 7. Photograph of insertion of the polyethylene tube with tapered cuff into a coronary ostium. The catheters are secured with a fine silk purse-string suture, and are removed just before the last portion of the aortic wall is closed by suture.

be better than in these early cases, because of greater experience in the proper technique of operation. In addition, such factors as the recent development of a more effective neutralizer of heparin, Polybrene®,[10] the perfection of methods of preventing air embolism,[11] an increased familiarity with the problems associated with the maintenance of coronary perfusion and now, new perfusion techniques (Fig. 7), will continue to improve our results.

SUMMARY

A review of our early experiences with 35 patients who had successful open heart surgery with the aid of the pump oxygenator, and who are now alive more than 18 months after surgery, is presented. Excellent grades of improvement persist in 34. These preliminary results have been encouraging, and have served as the impetus for the development of advanced techniques. The scope of the field of effective treatment, the facility and capability of the surgeon, and the safety of the patient have all shown constant improvement.

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Pure Ventricular Septal Defect and Ventricular Septal Defect with Pulmonic Stenosis

Hemodynamic and Clinical Changes Following Open Heart Surgery*

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SINCE THE DEVELOPMENT of open heart surgery with pump oxygenator techniques, repair of the ventricular septum has become feasible.[1,2] Furthermore, a combined lesion such as ventricular septal defect and pulmonic stenosis lends itself more easily to complete correction.

Many publications have dealt with the techniques, results and mortality involved in open heart surgery in a variety of congenital and acquired cardiac lesions.[3–6] Recent reports have aroused interest in the physiologic and clinical changes observed in a wide variety of congenital lesions.[7–9] It is the purpose of this communication to point out these physiologic and clinical changes in a group of patients with pure ventricular septal defect and ventricular septal defect with pulmonic stenosis, following satisfactory repair by open heart technique.

MATERIAL AND METHODS

Twelve patients who underwent open heart surgery for the correction of congenital cardiac defects were catheterized and clinically evaluated pre- and postoperatively. Five patients had ventricular septal defect with pulmonic stenosis and seven had ventricular septal defect alone.

Right heart catheterization was performed in the usual manner.[10] Blood gas analyses were performed by the method of Van Slyke and Neill.[11] Pressure recordings were made by the use of Statham P23D strain gauge manometers and recording was made by photo-oscillographic technique.†

Pulmonary vascular resistance was calculated from the formula of Gorlin and Gorlin, and right and left ventricular work as originally proposed.[12] Circulatory shunts were calculated according to the method of Van Dam and co-workers.[13]

Preoperative catheterization was performed a few days prior to surgery. Postoperative catheterization and clinical evaluation were performed 14 days to 1 year following surgical intervention.

Open heart surgery was performed with the use of the Sigmamotor pump and Freidland-Gemeinhardt oxygenator.[14]‡

As this communication is concerned only with the hemodynamic and clinical changes consequent to open heart surgery, this series is limited to patients in whom the shunt at the ventricular level was completely eliminated.

All patients but one were acyanotic, including those with ventricular septal defect and pulmonic stenosis. Of the patients with pure ventricular septal defect, two (P. J. and J. K.) had the defect repaired with implantation of Ivalon® sponge. In all but one (T.G.) of the other patients repair was made with interrupted

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‡ All patients operated upon by Dr. Charles P. Bailey, Hahnemann Hospital, Philadelphia, Pennsylvania.

TABLE I. Hemodynamic Data

Patient	O_2 consumption (cc/min)	Pulmonary A-V O_2 difference (vol %)	Systemic A-V O_2 difference (vol %)	Pulmonic blood flow (L/min)	Effective pulmonic blood flow (L/min)	Systemic blood flow (L/min)	Left-to-right shunt (L/min)	Right-to-left shunt (L/min)	Right atrium (mean) (mm Hg)	Right ventricle (mm Hg)
										Group I. Pure Ventricular
C. D.										
Preoperative	325	3.1	4.9	10.2	6.7	6.7	3.5	0	(−4)	40/−3
Postoperative	329	4.6	4.6	7.1	—	7.1	0	0	(−3)	17/0
M. K.										
Preoperative	205	2.6	4.5	5.6	3.7	4.6	1.9	0.9	(3)	50/8
Postoperative	197	7.0	7.0	3.3	—	3.3	0	0	(1)	27/0
C. J.										
Preoperative	169	1.8	5.0	9.4	—	3.3	6.1	0	(6)	42/6
Postoperative	264	4.7	4.7	5.6	—	5.6	0	0	(5)	36/7
P. J.										
Preoperative	—	0.8	3.6	—	—	—	—	—	(3)	62/3
Postoperative	—	3.4	3.4	—	—	—	0	0	(1)	34/0
D. D.										
Preoperative	—	1.7	6.3	—	—	—	—	—	(0)	30/1
Postoperative	—	4.8	4.8	—	—	—	0	0	(6)	25/5
T. G.										
Preoperative	192	2.0	4.2	6.6	3.4	4.6	3.2	1.2	(10)	48/13
Postoperative	214	3.1	3.1	3.3	3.3	3.3	0	0	(7)	65/10
J. K.										
Preoperative	—	—	—	—	—	—	—	—	(4)	26/0
Postoperative	—	2.7	2.7	—	—	—	0	0	(6)	27/0
										Group II. Ventricular Septal
H. V.										
Preoperative	201	—	—	7.0	3.3	3.8	3.2	0.5	(5)	44[b]/4 76/4
Postoperative	113	—	—	3.0	—	3.0	—	—	(2)	45/2
J. E.										
Preoperative	—	—	—	—	—	—	—	—	(1)	98/3
Postoperative	—	—	—	—	—	—	—	—	—	37/5
G. R.										
Preoperative	272	—	—	6.6	4.1	4.1	2.5	0	(5)	85/9
Postoperative	224	—	—	4.0	—	4.0	—	—	(0)	11/0
H. W.										
Preoperative	—	—	—	—	—	—	—	—	(4)	120/7
Postoperative	233	—	—	—	—	5.7	0	0	(−2)	25/0
J. J.										
Preoperative	—	—	—	—	—	—	—	—	(0)	100/−1
Postoperative	—	—	—	—	—	—	—	—	(4)	26/4

[a] Right ventricular wall sutured to septum around defect with interrupted nylon suture. [b] Infundibular.

Pulmonic Circulation				Systemic Circulation						
Pulmonary artery (mean) (mm Hg)	Pulmonary venous capillary (mean) (mm Hg)	Total pulmonary vascular resistance (dynes sec cm^{-5})	Effective right ventricular work (kg m/min)	Left ventricle (mm Hg)	Brachial artery (mm Hg)	Aorta (mm Hg)	Peripheral saturation (%)	Effective left ventricular work (kg m/min)	Heart rate	Type of closure
Septal Defect										
22/6 (12)	—	91	1.76	—	111/61 (83)	—	94	8.0	89	—
17/4 (9)	—	102	0.92	—	122/80 (90)	—	92	9.2	105	Suture
48/10 (29)	—	466	2.34	114/5	116/50 (80)	104/68	90	5.3	49	Suture
24/8 (14)	—	340	1.37	—	128/70 (83)	—	92	3.9	62	—
40/19 (30)	(11)	255	4.05	—	124/70 (79)	—	94	3.74	84	Suture
26/10 (16)	(6)	228	1.28	—	136/68 (87)	—	95	6.22	98	—
62/33 (48)	(8)	—	—	—	86/48 (59)	—	90	—	80	Ivalon sponge
34/10 (22)	(3)	—	—	—	80/45 (67)	—	95	—	80	—
23/5 (12)	(7)	—	—	—	—	—	90	—	88	Suture
24/8 (12)	(4)	—	—	—	—	—	89	—	92	—
40/18 (30)	(20)	364	2.82	—	90/35 (58)	—	83	5.40	100	[a]
29/16 (22)	(9)	535	1.05	—	128/60 (76)	—	89	3.72	120	[a]
26/10 (18)	(9)	—	—	83/8	—	80/58	93	—	90	Ivalon sponge
25/10 (17)	—	—	—	—	—	—	—	—	112	—
Defect and Pulmonic Stenosis										
23/6 (11)	(7)	46	1.1	—	111/64 (80)	—	91	4.4	84	Ivalon sponge
17/3 (12)	(6)	160	0.52	—	117/59 (84)	—	94	3.6	82	—
22/6 (14)	—	—	—	—	109/82 (90)	—	—	—	102	Suture
—	—	—	—	—	—	—	88	—	99	—
19/11 (14)	(4)	134	1.3	—	90/60 (73)	—	94	4.3	80	Suture
11/3 (9)	—	—	—	—	82/50 (66)	—	97	3.8	70	—
—	—	—	—	—	128/77 (93)	—	95	—	—	Ivalon sponge
16/4 (10)	—	—	—	—	116/52 (70)	—	92	—	—	—
16/0 (3)	(2)	—	—	—	106/70 (85)	—	73	—	140	Suture
18/8 (12)	—	—	—	—	87/55 (74)	—	91	—	110	—

nylon sutures; in T. G. the enlarged free wall of the right ventricle was sutured around the defect with interrupted nylon sutures.

It is of interest that one of these patients (M. K.) had multiple small defects in the interventricular septum. Eighteen interrupted sutures were required, each closing one small defect.

In the patients with ventricular septal defect and pulmonic stenosis, ventricular defects were closed in two (H. W. and H. V.) with the use of Ivalon sponge and in the other three with the use of interrupted nylon sutures. The pulmonic obstruction was relieved either by splitting the commissures under direct vision or by resection of the ridge or both.

For purposes of discussion we have divided our patients into two groups:

Group I: Seven patients with pure ventricular septal defect.

Group II: Five patients with ventricular septal defect and pulmonic stenosis.

For the clinical evaluation negative symptoms were not included. Patients were evaluated in terms of changes in symptomatology and physical findings as well as heart size, electrocardiogram and vectorcardiogram. Murmurs, heart sounds, and thrills were arbitrarily graded from 1 to 4. No attempt was made to grade clinical symptoms. They are merely noted if present. A 12-lead electrocardiogram and roentgenograms of the chest were taken before and after surgery.

Results

All pertinent physiologic and clinical data are summarized in Tables I and II, respectively. In all patients of both groups, the ventricular septal defect was completely closed as evidenced by the physiologic data.

Group I—Pure Ventricular Septal Defect

Hemodynamic Changes: The patients in this group were catheterized from 14 days to 1 year following open heart surgery. In all but two patients, the right ventricular and pulmonary arterial pressures were noted to fall following closure of the ventricular septal defect. In one patient (D. D.) with no fall in pressure, there was a normal pulmonary artery pressure preoperatively with no change following surgery. The other patient (T. G.) was the one in whom the free wall of the right ventricle was sutured around the defect. This apparently caused obstruction of the outflow tract of the right ventricle, as the postoperative catheterization data revealed a systolic pressure gradient between the main pulmonary artery and right ventricular outflow tract. The fall in the pulmonary artery pressure in this patient was probably due to the surgically created obstruction.

The greatest fall in pulmonary artery pressure was observed in patients with the highest pressure prior to surgery. Four of these patients showed evidence of moderate pulmonary hypertension during their first catheterization but the pressure fell to within normal limits after the defect was closed.

Because of the inability to obtain oxygen consumption, flow measurements in three patients were not available. In the other four the pulmonic blood flow and systemic blood flow fell in two, rose in one, and remained unchanged in one.

There was a marked reduction in the effective right ventricular work in all patients. The effective work of the left ventricle rose in three of the four patients in whom calculations were available after surgery. The total pulmonary vascular resistance was unchanged in two patients, fell in one and rose in another but remained within normal limits in the latter. There was no significant or consistent change in the peripheral arterial oxygen saturation except in one patient in whom there was a mild increase.

Clinical Findings: Three of the seven patients had no symptoms before or after surgery; of the other four, two had dyspnea, fatigue and paroxysmal nocturnal dyspnea; a third had dyspnea, fatigue and evidence of congestive heart failure; and the fourth had dyspnea and fatigue prior to surgery. No patient manifested any symptoms after surgery.

Three patients had a grade 3 systolic thrill which disappeared after surgery.

All patients had a grade 3 harsh systolic murmur over the left sternal border radiating in some patients to other cardiac areas (e.g., mitral, aortic, pulmonic, and tricuspid areas). The murmur disappeared after surgery in two patients, decreased in intensity in four, and remained unchanged in one.

The second pulmonic sound which was accentuated in all but two patients prior to surgery became either normal or split following

TABLE II
Clinical Data Before and After Surgery

Patient		Dyspnea	Fatigue	Thrill	Second pulmonic sound	Murmur	Post-operative period
		Group I. Pure Ventricular Septal Defect					
C. D.	Preoperative	0	0	3+ (systolic)	2+	3+ systolic	2 months
	Postoperative	0	0	0	Normal	0	
M. K.	Preoperative	+	+	0	2+	3+ rough systolic	3 months
	Postoperative	0	0	0	Split	0	
C. J.	Preoperative	0	0	0	2+	3+ systolic	12 months
	Postoperative	0	0	0	Normal	1+ systolic	
P. J.	Preoperative	+	+	3+ (systolic)	2+	3+ systolic	2 weeks
	Postoperative	0	0	0	1+	1+ systolic	
D. D.	Preoperative	+	+	0	Normal	3+ systolic	2 months
	Postoperative	0	0	0	Normal	1+ systolic	
T. G.	Preoperative	+	+	3+ (systolic)	Normal	3+ systolic and 2+ early diastolic	1 month
	Postoperative	0	0	0	Normal	3+ systolic	
J. K.	Preoperative	0	0	0	2+ split	4+ systolic	2½ months
	Postoperative	0	0	0	Normal	2+ systolic	
		Group II. Ventricular Septal Defect with Pulmonic Stenosis					
H. V.	Preoperative	+	+	3 to 4+	Decreased	4+ systolic	5 months
	Postoperative	0	0	0	Normal	2+ systolic	
J. E.	Preoperative	0	+	0	Normal	2 to 3+ systolic	5 months
	Postoperative	0	0	0	Normal	2+	
G. R.	Preoperative	+	+	0	Decreased	3 to 4+ systolic	2 months
	Postoperative	0	0	0	Split	1 to 2+ systolic	
H. W.	Preoperative	+	0	0	Normal	3+ systolic	6 months
	Postoperative	0	0	0	Normal	1+ systolic	
J. J.	Preoperative	+	+	0	Decreased	3+ systolic	6 months
	Postoperative	0	0	0	Normal	1+ systolic	

surgery except in one patient in whom its intensity diminished from 2+ to 1+.

It is of interest to note that in all patients a complete right bundle branch block was present in the electrocardiogram and vectorcardiogram following repair of the ventricular septal defect.[15]

Radiographic films were evaluated in six of the patients in this group. They were unavailable in one patient (T. G.). The heart size was enlarged from 1+ to 3+ (on the basis of 1+ to 4+) preoperatively, this generally being the reflection of an increase of right ventricular mass. The pulmonary vascular markings were also increased from 1+ to 3+. Postoperatively the heart size was slightly increased in all patients.

GROUP II—VENTRICULAR SEPTAL DEFECT WITH PULMONIC STENOSIS

Hemodynamic Changes: All patients in this group were catheterized from two to six months following surgery. In general, the pulmonary artery pressure of these patients changed very little after surgery. However, the right ventricular pressure was markedly reduced and fell to nearly normal limits after surgery. The systolic pressure gradient between the right ventricle and pulmonary artery was greatly reduced; indeed in one patient (G. R.) no appreciable gradient could be demonstrated. The right ventricular end diastolic and right atrial pressures remained within normal limits following surgery.

In two patients (H. V. and J. J.) there was an infundibular ridge as well as a valvular pulmonic stenosis; in all the others valvular stenosis alone was present.

Again, because of technical difficulties, flow measurements were made only in two of the five patients. The pulmonic blood flow showed a significant fall in both of these patients. There was no significant change in the systemic blood flow.

The effective work of the left ventricle was slightly less in both patients following surgical correction. There was no significant change in the total pulmonary vascular resistance. Also, there was no significant change in the peripheral arterial saturation, except in one patient in whom there was a marked increase. Another patient (J. E.) had a peripheral arterial unsaturation of 88 per cent, possibly due to use of general anesthesia.

Clinical Findings: Clinically, all of these patients had at least one symptom. Three had dyspnea and fatigue, a fourth had only fatigue and the fifth had dyspnea alone. Only one patient had a systolic thrill and this disappeared after surgery. The second pulmonic sound was markedly decreased in three patients and normal in two preoperatively. Following surgery the second pulmonic sound became normal in all patients except in one (G. R.) in whom it was split. A systolic murmur along the left sternal border and pulmonic area varying in intensity from 1+ to 4+ was present in all patients prior to surgery. This systolic murmur persisted in all patients following surgery, but was decreased in intensity. Only one patient (J. J.) gave a history of cyanosis prior to surgery. This disappeared following surgery.

As noted in group I, in all patients in this group complete right bundle branch block developed in the electrocardiogram and vectorcardiogram after correction of the lesions. Radiographic films were evaluated in only three patients in this group, being unavailable in two (H. V. and J. E.). The heart size was increased slightly in all patients. The pulmonary artery was increased in size in only one patient while the pulmonary vascular markings were normal in all. The increased size of the pulmonary artery was due to poststenotic dilatation. The pulmonary vascular markings were also unchanged after surgery. Again, in general there was a slight increase in the heart size postoperatively.

Comments

Pulmonary Artery Pressure: Elevation in pulmonary arterial pressure in ventricular septal defect is not only due to changes in pulmonary vascular resistance as has been previously stressed,[16] but also may be due to an increased pulmonic blood flow. Sones[7] in his series has indicated that severe pulmonary hypertension alone is not a contraindication to closure of ventricular septal defect. In his series two patients with severe pulmonary hypertension showed a marked fall in pulmonary pressure following surgical correction.

This was also true in one patient in our group in whom the pulmonary pressure returned to nearly normal limits within 14 days following closure of his ventricular defect. It is of further interest that in this patient as well as in Sones' series, the pulmonary pressure was 70 per cent or more of the systemic pressure. This would tend to indicate that the selection of patients for repair of ventricular septal defect on the basis of the pulmonary pressure being 70 per cent or less of the systemic pressure as suggested by Lillehei[8] should be tempered so as to determine whether hypertension is caused by organic changes or increased flow.

Pulmonary Blood Flow: Because it was not possible to measure the pulmonic blood flow in all patients the pulmonic and systemic arteriovenous oxygen differences were used as indices of changes in pulmonic blood flow. It can be seen from Table I that the pulmonary arteriovenous oxygen difference was the same as the systemic arteriovenous difference in all patients postoperatively, indicating a reduction in pulmonic blood flow. Thus, the fall in pulmonary pressure in all patients in our series could be explained on the basis of reduction of flow.

Right Ventricular Pressure and Work: In patients with ventricular septal defect and pulmonic stenosis, a fall in right ventricular pressure is to be expected following relief of the obstruction in either the pulmonic valve or right ventricular outflow tract or both. Therefore, the fall in pressure is primarily one of relief of this obstruction and only secondarily due to reduction in pulmonic blood flow. In all patients in our series with this combination there was no calculated right-to-left shunt. That the work of the right ventricle fell in both groups I and II following surgery is a reflection both of decreased resistance and lowering of pulmonic blood flow.

Changes in Clinical Status: In general, in both groups the improved hemodynamics revealed by cardiac catheterization are reflected in the improved clinical status of the patients.

As has been reported by others there seems to be a slight increase in heart size in both groups following surgery.[7-9,15] We could not find any satisfactory explanation for this phenomenon.

All patients in this series were noted to have a complete right bundle branch block following surgery. This has also been reported by others.[7-9] Truex and Bishof[17] have shown that the common bundle and the right and left branches are located in the posteroinferior margin of the ventricular septal defect. During the time of surgical repair either by direct suture or by Ivalon implantation, sutures were placed into the immediate area and right bundle branch block ensued. Therefore, proper precaution should be taken to avoid that area in so far as is possible, as has been suggested by Kirklin and his associates.[1]

We cannot demonstrate any physiologic or clinical differences between direct closure with nylon suture and/or placement of Ivalon sponges into the interventricular septum.

The persistence of the systolic murmur in the patients in group II possibly reflects a residual minimal stenosis at the pulmonic area, as can be seen in Table II.

Tetralogy of Fallot: It has been previously suggested that the term tetralogy of Fallot is physiologically inaccurate.[18,19] In this condition the two anatomic lesions are: (1) the valvular or infundibular stenosis and (2) the interventricular septal defect. The right ventricular hypertrophy is the result of the stenotic component. The fact that closure of the interventricular septal defect in our series had no adverse effect on the patients precludes the possibility of true anatomic overriding of the aorta.

Selection of Patients for Surgery: Selection of patients for surgery cannot be characterized on the basis of any single physiologic or clinical entity. The age of the patients in our series seems to have had no bearing on the operative results. As suggested by Sones,[7] pulmonary hypertension plus a low pulmonic blood flow would seem to be one definite contraindication, along with the position of the aorta and size of the ventricular septal defect. Likewise, a poor clinical state or decreased myocardial reserve demonstrated by repeated attacks of congestive heart failure, would seem to influence selection.

According to our data we can say that a patient with a pure ventricular septal defect or ventricular septal defect with pulmonic stenosis, when well selected and preoperatively well prepared, can tolerate closure of the ventricular septal defect. The right bundle branch block which takes place postoperatively perhaps is of no physiologic importance.

In contrast, we noted that in patients with a marked elevation in pulmonary artery pressure the mortality is very high, a fact which has been previously observed by several other investigators.[6,20] Although we recognize that our present technique for open heart surgery is not an ideal one, with the refinement of these techniques and increased safety factors, open heart surgery will be the procedure of choice for the majority of patients with these lesions, as well as for many other types of congenital heart disease.

Summary

(1) Pre- and postoperative right heart catheterization and clinical evaluation were performed in twelve patients who underwent open heart surgery. Seven had pure ventricular septal defects while five had ventricular septal defect and pulmonic stenosis.

(2) In all patients in this series the pulmonary artery and right ventricular pressures fell postoperatively, as well as the pulmonic blood flow, right ventricular work and the total pulmonary vascular resistance, while the left ventricular work was increased. Physiologically and clinically, all patients were improved in this selected series.

(3) In both groups a complete right bundle branch block was observed in the electrocardiogram and vectorcardiogram postoperatively.

(4) Open heart surgery when indicated is the treatment of choice for patients with either pure ventricular septal defect or ventricular septal defect with pulmonic stenosis.

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Postoperative Physiopathologic Results in Mitral Stenosis*

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WE PRESENT in this report the early and late physiopathologic changes which occur in patients with mitral stenosis after mitral commissurotomy. From our operative case material we chose 200 patients whose course could be followed for a long period after commissurotomy, and who had been catheterized preoperatively. In 28 of them we recorded the pressure pulses of the left heart during surgical intervention. In 47 of them we recorded the pressure pulses of the right heart and other hemodynamic data at intervals of two to five months, one year, two years and three years after commissurotomy.

POSTOPERATIVE PERICARDITIS

The physiopathologic modifications (fever, chest pains, tachycardia, left pleural reaction with or without effusion, and pericarditis) which appear during the early days after operation are a consequence of the surgical intervention itself and are not provoked by rheumatic reactivation. Ninety per cent of the patients had marked signs of pericarditis with a rub and electrocardiographic alterations (negative T wave in all leads and, more rarely, negative T_a, especially in the limb leads); radiographic signs of pericarditis were less frequent. In all cases the electrocardiogram and x-ray findings later returned to normal.

POSTPERICARDIOTOMY SYNDROME

The syndrome called "postcommissurotomy" by Soloff *et al.*[1,2] ought rather to be called "postpericardiotomy" because it is not necessary to perform the commissurotomy for it to appear. We observed the symptoms of this syndrome in two patients on whom we carried out only a valvular exploration because we found predominant mitral insufficiency existed. We must not confuse this postpericardiotomy syndrome with the chest pains, pericarditis, pleuritis, and fever which follow immediately after the operation and which disappear in a few days. If in a week, fortnight, two or three months after the operation fever, chest pains, and sometimes articular pains recur and the electrocardiogram, after becoming normal, shows signs of pericarditis with or without pleural reaction, the erythrocyte sedimentation rate is high, the C-reactive protein is positive, and the antistreptolysin 0 titer is high, then we are dealing with the postpericardiotomy syndrome or as it is generally called, the postcommissurotomy syndrome.

In two patients the syndrome began early, within the period of the usual reactions to the operation. This made interpretation difficult, but the marked signs of pericarditis in the electrocardiogram and the disappearence of all symptoms when prednisolone was administered showed it was the postpericardiotomy syndrome due to rheumatic reactivation following the operation.

In this series of 200 patients on whom commissurotomy was performed 25 (12.5 per cent) showed this syndrome. In two patients the syndrome appeared within two weeks after the operation, in the remaining 23 patients, one, two, or three months afterwards. It was always accompanied by electrocardiographic signs of pericarditis. The syndrome appeared only once in each patient, except in four patients in whom it appeared three or four times with asymptomatic intervals between the recurrences. On each reappearance of the symptoms

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the electrocardiogram showed diagnostic signs of pericarditis.

Late Rheumatic Reactivation: We found no relation between the appearance of the postoperative syndrome, late rheumatic reactivation, or the appearance of the auricular appendage. The histologic examination of the auricular appendage of the 25 patients with postoperative syndrome showed no Aschoff bodies or any other histologic signs of rheumatic reactivation.

We observed late rheumatic reactivation in six patients, eight months and one year after the operation. Three of these patients had no rheumatic fever for two, four, and five years, respectively, before the operation. In spite of these attacks of rheumatic fever the clinical improvement was maintained. We observed two cases in which stenosis recurred through late rheumatic reactivation.

Electrolytic Alterations: Appearing in the first few days, these consisted of hyponatremia and hyperpotassemia but they disappeared rapidly without causing trouble.

Physiopathologic Results

The physiopathologic results of mitral commissurotomy were studied by cardiac catheterization, phonocardiogram, electrocardiogram, vectorcardiogram, electrokymogram, ballistocardiogram, roentgenogram, pulmonary angiocardiography, and pulmonary function tests, and by observing the improvement in symptoms, especially pulmonary symptoms: dyspnea, pulmonary edema, hemoptysis, cyanosis.

Preoperative Left Heart Catheterization

Theoretically, the best method of appreciating hemodynamically the efficiency of commissurotomy would be to determine the left atrial and ventricular pressures at the time of operation, immediately before and immediately after commissurotomy. This method has been used by some cardiologists.[3–9] We carried it out in 28 patients, and we show the data of 20 (Table I). In 11 patients we took the atrial and ventricular pressures simultaneously so that we were able to determine the diastolic gradient more accurately (Fig. 1). In the others it was not possible to take simultaneous readings. We also recorded the pulmonary pressures.

We divided these 20 patients into three groups according to the magnitude of decrease in mean atrial pressure. Table I lists ten patients with a decrease in the mean pressure of 5 mm Hg or

TABLE I

Left Heart Catheterization Data at Operation

Case No.	Age (yr)	Remarks		Pressure (mm Hg)															
				Left atrium						Left ventricle			Pulmonary artery			Aorta			
				Maximum	Minimum	Mean	End diastolic pressure	A wave	C wave	V wave	S.	D.	Gradient	S.	D.	Mean	S.	D.	Mean
		A. Decrease in Mean Atrial Pressure of 5 mm Hg or More																	
1	37	Decrease of regurgitation after commissurotomy	Pre	34	21	28	—	Flutter	28	34	150	20	—	55	29	40	—	—	—
			Post	25	14	21	—	Flutter	24	25	137	29	—	—	—	—	—	—	—
2	28		Pre	43	32	37	—	36	42	43	—	—	—	107	66	78	—	—	—
			Post	35	12	27	—	27	35	35	—	—	—	82	42	62	—	—	—
3	26		Pre	32	15	25	—	30	25	32	96	13	—	46	21	—	—	—	—
			Post	24	10	17	—	24	24	22	100	10	—	46	25	32	—	—	—
4	32		Pre	31	18	23	25	23	28	31	105	17	8	49	19	33	89	53	67
			Post	22	11	15	21	18	21	22	130	17	4	42	12	27	—	—	—
5	17	Incomplete commissurotomy	Pre	56	30	48	44	50	56	48	154	0	44	85	42	58	—	—	—
			Post	36	14	26	21	Flutter	36	36	72	5	16	—	—	—	—	—	—

6	30		Pre	26	15	20	—	21	26	20	—	—	—	92	48	70	—	—	—
			Post	13	6	8	—	8	13	9	—	—	—	—	—	—	—	—	—
7	25		Pre	45	19	30	35	45	42	35	115	0	35	116	53	78	—	—	—
			Post	35	18	25	28	32	35	33	125	10	18	—	—	—	—	—	—
8	22		Pre	58	29	40	—	40	58	47	—	—	—	122	75	92	—	—	—
			Post	45	18	33	—	29	43	45	—	—	—	—	—	—	—	—	—
9	39	Incomplete commissurotomy	Pre	38	26	30	23	—	30	38	90	15	8	45	23	31	—	—	—
			Post	37	18	24	—	—	26	32	—	—	—	—	—	—	—	—	—
10	25		Pre	34	10	26	16	34	26	26	96	6	10	28	16	20	—	—	—
			Post	16	8	11	10	16	14	14	102	10	—	28	16	20	—	—	—
								B. Decrease in Mean Atrial Pressure of Less Than 5 mm Hg											
11	23		Pre	30	13	21	—	30	19	23	121	18	—	46	24	33	—	—	—
			Post	23	11	19	—	23	c + v plateau 20		78	24	—	38	18	31	94	39	59
12	17		Pre	31	17	19	17	31	19	24	153	9	8	24	13	18	—	—	—
			Post	23	12	15	11	23	16	19	161	9	2	—	—	—	—	—	—
13	33		Pre	44	30	37	—	40	44	41	—	—	—	82	45	63	—	—	—
			Post	49	21	35	—	35	49	40	—	—	—	—	—	—	—	—	—
14	46		Pre	40	15	27	18	22	33	40	121	14	4	58	26	34	—	—	—
			Post	32	15	26	—	28	32	32	103	20	—	61	33	44	—	—	—
15	37		Pre	22	13	17	18	18	22	20	103	7	11	55	28	42	73	50	65
			Post	20	10	14	14	14	18	20	122	14	0	—	—	—	106	69	90
16	22	Slight regurgitation after commissurotomy	Pre	20	10	15	18	20	19	16	80	8	10	40	20	25	—	—	—
			Post	16	7	13	12	16	15	14	108	12	0	31	15	20	—	—	—
17	25		Pre	24	14	18	—	24	24	20	102	10	—	52	30	40	—	—	—
			Post	20	14	16	—	16	18	20	98	14	—	27	15	20	—	—	—
								C. Variable Changes in Mean Atrial Pressure											
18	26		Pre	17	12	14	—	12	12	17	101	11	—	23	13	—	—	—	—
			Post	18	11	14	—	18	16	18	—	—	—	23	13	—	—	—	—
19	21	Slight regurgitation after commissurotomy	Pre	26	18	21	24	24	25	21	—	10	14	31	19	23	—	—	—
			Post	30	24	29	28	25	30	25	108	19	9	26	17	20	—	—	—
20	39		Pre	22	8	13	18	20	22	13	135	2	16	32	13	22	135	82	113
			Post	31	18	25	30	30	31	31	150	13	17	32	16	23	—	—	—

NOTE: Pre = preoperative.
Post = postoperative.

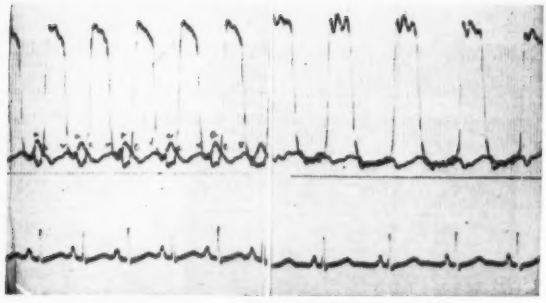


FIG. 1. Simultaneous left atrial and ventricular pressure pulses taken during surgery. *Left*, before mitral commissurotomy. *Right*, after mitral commissurotomy.

more, seven with a more moderate decrease—1 to 4 mm Hg; and three with variable results.

Group I: In these ten cases in which it was possible to establish the gradient there was always a marked decrease, but there was only a return to normal in two cases. A decrease in pulmonary pressure also occurred but there was no parallelism between this and the decrease in the gradient. In all these cases the patients showed good clinical progress (see Table I).

Group II: These seven cases in which there was a smaller decrease in atrial pressure, were cases of pure mitral stenosis. In those in which it was possible to determine the gradient we noted a return to normal. The influence on pulmonary pressure was small. In all but one case (Case 15) the clinical results were excellent. In this case the patient's condition remained stationary.

Group III: In one case of severe stenosis (Case 18), partially opened digitally, the mean atrial pressure did not change. In a case of pure stenosis (Case 19), there was postoperative regurgitation; the mean atrial pressure went up and the pulmonary pressure went down slightly. In a case of predominant stenosis (Case 20), there was an increase in mean atrial pressure after anterior commissurotomy. In all these cases, the clinical condition of the patient remained stationary.

Significance of the Results: How should we explain these discrepancies? As Dickens *et al.*[10] have already stated, the gradient of atrial and ventricular pressures is by itself insufficient to evaluate the results of commissurotomy. If there is an appreciable increase in flow across the mitral valve accompanied by a fall in the gradient then results should be excellent. A small decrease in the gradient gives no indica-

TABLE II
Right Heart Catheterization Data (Less Than Six Months Postoperative)

Case No.	Age (yr)		Mitral valve flow cc/sec		Pressure (mm Hg)																Cardiac Index (L./min./m²)	
					Pulmonary artery wedge						Pulmonary artery				Right ventricle			Right atrium				
					Rest			Exercise			Rest			Exercise								
			Rest	Exercise	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	Rest	Exercise
											A. Excellent Clinical Results												
21	59	Preoperative	165	—	39	18	24	50	25	30	56	39	46	70	42	56	—	—	—	—	—	4	3.5	3.7
		Postoperative	204	—	18	5	9	20	8	15	40	20	30	45	20	33	45	7	—	—	—	4	3.5	3.1
22	37	Preoperative	146	—	35	12	22	50	25	37	75	25	41	92	27	55	80	13	—	—	—	17	2.9	2.7
		Postoperative	120	—	18	6	11	25	10	14	25	6	13	32	12	19	27	0	—	—	—	2	2.8	3.4
23	51	Preoperative	170	—	56	25	35	66	29	45	75	38	60	80	40	65	75	12	—	—	—	9	3.2	3.5
		Postoperative	120	—	30	15	25	32	20	28	55	25	42	57	30	45	55	5	—	—	—	10	2.4	2.5
24	27	Preoperative	150	—	45	38	41	—	—	—	80	65	55	—	—	—	80	5	21	9	16	16	3.3	—
		Postoperative	—	—	47	15	25	—	—	—	62	25	—	—	—	—	60	6	8	0	5	5	—	—
25	27	Preoperative	216	—	22	17	20	—	—	—	65	30	40	—	—	—	66	10	12	6	10	10	4.8	—
		Postoperative	—	—	15	3	7	—	—	—	50	12	—	50	35	—	50	5	4	0	3	3	—	—
14	46	Preoperative	98.6	—	23	17	19	—	—	—	47	23	33	68	42	50	47	4	3	− 1	+ 1	1	1.8	—
		Postoperative	131.8	—	—	—	13	—	—	20	28	14	21	38	23	29	29	5	7	2	4	4	2.4	—
6	30	Preoperative	87	—	—	—	—	—	—	—	145	65	90	—	—	—	155	15	20	10	15	15	2.1	—
		Postoperative	104	—	—	—	—	—	—	—	51	20	33	—	—	—	51	2	4	1	2	2	2.2	—

26	28	Preoperative	116.6	—	—	—	29	—	—	—	90	33	60	—	—	—	89	3	—	—	0	2.6	—
		Postoperative	131	—	—	—	13	—	—	—	38	18	25	52	30	—	40	0	—	—	− 2	3.1	—
7	25	Preoperative	91	—	125	40	77	—	—	—	130	40	83	—	—	—	140	2	7	1	5	2.3	—
		Postoperative	146	—	—	—	14	—	—	—	48	22	33	—	—	—	46	2	3	1	2	2.9	—
27	28	Preoperative	164	—	41	25	32	—	—	—	58	37	46	—	—	—	60	9	10	5	8	3.6	—
		Postoperative	178	—	23	10	15	18	—	—	29	18	23	74	24	53	42	0	2	− 2	0	4.2	—
28	16	Preoperative	—	—	—	—	34	—	—	—	72	36	50	110	50	94	70	6	—	—	0	2.5	3.1
		Postoperative	130	—	18	13	15	28	15	25	40	18	24	66	28	42	40	0	12	− 3	6	2.3	3
29	51	Preoperative	—	—	29	25	27	—	—	—	38	30	34	64	56	60	40	3	15	12	13	2.2	—
		Postoperative	163	—	—	—	12	30	18	25	32	15	24	48	20	35	35	3	4	0	2	2.7	—
30	30	Preoperative	114	—	20	10	15	33	16	25	30	12	21	55	18	32	45	2	10	2	7	2.1	2.9
		Postoperative	161	—	14	8	10	29	11	18	30	11	17	44	11	23	31	3	8	2.5	—	3	—
16	22	Preoperative	99.8	—	42	28	35	—	—	43	53	32	43	77	48	60	52	8	—	—	5	2.1	—
		Postoperative	—	—	—	—	12	—	—	—	35	13	18	45	18	30	—	—	—	—	2	—	—
31	40	Preoperative	161.7	—	—	—	—	—	—	—	90	55	65	—	—	—	90	10	—	—	15	3.2	—
		Postoperative	113.6	—	—	—	—	—	—	—	48	33	40	65	53	60	46	9	6	3	5	2.3	—
		B. Good Clinical Results																					
4	39	Preoperative	107	—	45	25	35	58	28	42	88	43	67	100	48	70	90	− 5	—	—	4	2	2.5
		Postoperative	60	—	30	18	25	33	20	28	50	25	42	55	37	46	50	4	—	—	8	—	—
8	22	Preoperative	102	—	25	20	22	37	25	33	60	35	45	75	40	55	60	5	9	5	—	—	—
		Postoperative	—	—	34	18	24	29	13	23	47	23	36	58	38	46	52	7	—	—	6	—	—
32	27	Preoperative	290	—	39	29	33	—	—	49	64	31	42	80	46	57	64	3	—	—	—	2.7	—
		Postoperative	213	—	—	—	23	—	—	—	40	24	31	57	28	40	47	13	—	—	9	2.83	3.51
33	20	Preoperative	155	—	—	—	26	—	—	51	50	29	38	85	34	58	50	5	10	5	7	2.8	—
		Postoperative	190	—	27	16	21	26	21	23	41	22	30	68	35	46	43	− 1	8	0	5	2.7	2.9
34	26	Preoperative	152	—	18	5	10	—	—	—	40	10	20	100	40	60	40	0	6	2	4	2.9	—
		Postoperative	147	160	—	—	—	—	—	—	30	15	18	63	23	43	40	3	5	− 2	0	2.9	3.1
		C. Variable Clinical Results																					
35	46	Preoperative	135	—	—	—	28	—	—	—	—	—	44	—	—	—	—	—	—	—	—	2.4	2.8
		Postoperative	142	—	42	20	25	50	25	30	65	25	42	75	39	48	65	− 1	—	—	− 3	3.2	3.2
36	37	Preoperative	188	—	32	15	24	30	15	24	38	14	27	40	12	25	46	0	—	—	2	3	—
		Postoperative	133	—	32.5	10	20	37.5	15	24	37.5	15	27.5	37.5	15	27	37.5	0	—	—	4	2.7	2.6
37	42	Preoperative	142	—	20	10	13	30	15	21	36	10	24	40	16	28	36	2	—	—		3.8	—
		Postoperative	150	—	18	12	15	23	18	20	33	18	25	34	28	32	33	5	—	—	1	3.0	3.4
38	42	Preoperative	—	—	32	20	28	—	—	—	50	20	30	85	38	62	55	7	10	2	5	3.6	4
		Postoperative	159	—	—	—	25	—	—	—	47	24	36	83	41	55	46	1	12.6	3	—	2.9	—
39	47	Preoperative	—	—	40	22	30	—	—	—	75	35	43	—	—	—	75	10	15	6	9	—	—
		Postoperative	—	—	27	16	20	35	20	30	56	28	40	72	45	56	55	7	10	6	8	1.7	2.4
40	27	Preoperative	148	—	30	20	25	37	30	34	40	25	32	55	30	40	40	8	—	—	9	2.9	—
		Postoperative	164	—	32	8	20	—	—	—	37	20	34	—	—	—	54	6	3	0.5	1.5	3.2	—
41	34	Preoperative	219	—	25	18	20	—	—	—	40	25	30	—	—	—	45	10	10	5	7	4.34	—
		Postoperative	115	—	38	18	27	—	—	—	60	38	50	—	—	—	65	5	8	3	5	2.1	—
42	18	Preoperative	152	—	—	—	20	—	—	24	45	26	34	67	23	55	45	2	3	0.5	2	2.8	—
		Postoperative	155	—	—	—	—	—	—	—	44	24	30	70	40	50	44	2	—	—	2	2.9	—

tion as to the postoperative hemodynamic alterations unless the accompanying change in mitral valve flow is also known. Generally, pulmonary artery pressure decreases considerably along with the fall in the gradient, increase in mitral area and increase in valve flow. Pulmonary pressure may, however, fall only slightly immediately after the operation in spite of the fall in the gradient and even in spite of the increase in mitral area and flow. If there are severe organic arteriolar lesions the pulmonary pressure may be maintained or fall only slightly at the operation. On the other hand, the fall in pressure of the left atrium is generally not very marked immediately after the commissurotomy.

There may be some decrease in diastolic gradient due to an increase in the diastolic pressure of the ventricle resulting from better filling; the atrial pressure is thus unchanged. Therefore, the fall in atrial pressure and change in diastolic gradient by themselves cannot serve as a measure of the physiopathologic alterations. Nor does there always exist a parallelism between fall in atrial pressure and clinical improvement. In some patients the atrial pressure recorded immediately after the operation showed only a slight fall which gave no indication of the subsequent clinical improvement.

POSTOPERATIVE RIGHT HEART CATHETERIZATION

Of 45 patients on whom we carried out postoperative catheterization, 28 were catheterized within the first six months, the remaining 17 at varying periods after the operation—one year, two years, three years. We have divided them into two groups—less than six months and more than six months.

Cardiac Catheterization Less Than Six Months Postoperative: We have further subdivided the first group into three subgroups (Table II). Fifteen patients had excellent clinical results; in most of them there was a very marked decrease in pulmonary artery and pulmonary capillary pressures. Five patients had a good or smaller decrease in pulmonary artery and pulmonary capillary pressures but with good clinical results. Eight patients with varying clinical results showed only slightly decreased or even increased pressures.

Opinion is unanimous that a hemodynamic examination carried out a few months afterwards does not always show the improvement which will be encountered a year or more after the operation.[11,12] Our own experience confirms this conclusion.[13] Nevertheless Group A in Table II shows large decreases in pulmonary artery and capillary pressures even to the extent of complete normalization. There is also an increase in mitral valve flow and area. For example, in Cases 6, 7, and 26, the preoperative pulmonary pressures were, respectively, 145/65 (mean 90), 130/40 (mean 83) and 90/30 mm Hg (mean 60), and after the operation they fell, respectively, to 51/20 (mean 33), 48/22 (mean 33) and 33/18 mm Hg (mean 25). The mitral valve flow and mitral area increased.

Although in the other cases the decreases in pressures were not so notable, all showed marked clinical improvement. The patients in Group B (Table II) all showed good clinical improvement. In one case (Case 25) the pulmonary pressure became normal; in the others the mean pressure fell from 67 to 42, from 45 to 36, from 42 to 41, and from 38 to 30 mm Hg. There was no corresponding increase in mitral flow in the latter four cases. This was due to two circumstances: incomplete commissurotomy in two cases and slight postoperative regurgitation in the other two.

In the eight cases in Group C (Table II) there have been slight decreases, no change, or increase in the pressures. The clinical results in these cases varied, without correlation with the alterations in pressures. For example, in Case 39 in which there was no clinical change, the mean pulmonary capillary pressure fell from 30 to 20 mm Hg and the mean pulmonary pressure fell slightly from 43 to 40 mm Hg. On the other hand there were cases like Case 35 in which there was good clinical improvement but the pressures have remained practically unchanged. In this case there was preoperative regurgitation. In Case 41 there was worsening of the patient's condition due to postoperative regurgitation in spite of incomplete commissurotomy.

Cardiac Catheterization Six Months or more Postoperative: The second group, composed of the 17 cases in which catheterization was made at a later period, is subdivided into three groups (Table III). There were nine patients with very marked decrease in or normalization of pulmonary artery and capillary pressures and excellent clinical results (Group A). In Case 44, for example, we see a systolic pulmonary pressure which falls from 120 to 40 mm Hg and a mean pulmonary pressure which falls from 74 to 35 mm Hg. The mitral output, mitral area and

TABLE III

Right Heart Catherization Data (More Than Six Months Postoperative)

Case No.	Age (yr.)		Mitral valve flow (cc/sec)		Pressure (mm/Hg)																	Cardiac Index (L/min/m²)	
					Pulmonary artery wedge						Pulmonary artery						Right ventricle		Right atrium				
					Rest			Exercise			Rest			Exercise									
			Rest	Exercise	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	S.	D.	Mean	S.	D.	S.	D.	Mean	Rest	Exercise
										A. Marked Decrease in Pulmonary Pressure													
43	27	Preoperative	129	—	—	—	39	—	—	—	111	53	80	—	—	—	111	5	13	5	10	2.4	—
		Postoperative	—	—	—	—	—	—	—	—	52	29	40	73	60	65	—	—	—	—	3	3.3	—
44	16	Preoperative	—	—	43	16	26	—	—	—	120	45	74	—	—	—	120	4.5	—	—	4	—	—
		Postoperative	210	—	22	13	20	—	—	20	40	20	35	85	45	60	47	−2	7	0	3	3.7	4.5
5	32	Preoperative	144	—	40	20	29	50	26	36	64	24	48	95	36	60	70	0	—	—	3	3.2	—
		Postoperative	183	—	20	10	13	28	15	20	30	10	17	55	30	40	32	0	8	0	5	3.6	3.75
45	24	Preoperative	179	—	40	32	37	37	29	32	80	40	58	100	42	63	95	−3	5	−2	0	3.2	3.1
		Postoperative	134	—	—	—	—	35	8	14	28	8	17	50	20	32	30	0	6	0	2	2.8	3.2
46	27	Preoperative	128	—	35	18	25	40	18	25	38	18	27	45	18	30	40	0-5	8	2	4	2.6	—
		Postoperative	146	—	16	10	12	32	13	20	24	8	15	42	18	28	30	0-6	6	2	4	3.2	3.1
47	21	Preoperative	119	—	40	28	32	45	35	40	110	46	80	160	75	120	108	0-5	8	3	5	2	3.7
		Postoperative	164	—	25	13	18	60	35	43	40	23	30	—	—	—	50	0	4	−2	2	3.1	—
48	31	Preoperative	114	—	35	20	26	—	—	—	43	19	28	—	—	—	48	2	—	—	7	2.4	—
		Postoperative	121	—	—	—	16	—	—	28	30	11	20	55	28	41	45	0	7	−3	6	2.5	—
49	56	Preoperative	115	—	35	15	20	—	—	30	50	20	30	75	30	45	55	5.5	—	—	10	2.6	—
		Postoperative	98	—	19	12	15	—	—	24	40	17	26	78	32	48	53	11	—	—	10	2.6	—
50	46	Preoperative	88	—	—	—	27	—	—	—	71	31	48	—	—	—	71	5	9	5	7	2.07	—
		Postoperative	107	—	37	22	33	—	—	—	52	32	40	80	39	54	58	0	9	6	7	2.1	2.35
										B. Pulmonary Pressure Same or Increased													
51	12	Preoperative	90	—	28	18	22	40	22	30	40	18	30	55	22	36	50	2	—	—	4	2.7	—
		Postoperative	—	—	24	15	18	—	—	33	37	23	28	56	26	35	38	5	7	3	—	2.1	—
52	37	Preoperative	120	—	22	8	14	38	15	21	31	11	23	44	15	25	34	0	—	—	2	3.2	—
		Postoperative	158	—	35	20	30	—	—	—	50	35	43	—	—	—	50	7	8	6	7	2.75	—
53	11	Preoperative	95	—	25	8	18	—	—	—	30	15	20	47	24	32	37	2	8	0	2	3.4	—
		Postoperative	200	—	26	17	22	33	23	28	29	16	23	58	34	46	40	0	6	2	4	4.07	6.45
										C. Divergent Hemodynamic and Clinical Results													
54	38	Preoperative	128	—	60	25	37	—	—	—	80	37	60	89	40	66	85	45	—	—	8	3.1	3.7
		Postoperative	160	—	30	15	20	—	—	—	60	22	40	—	—	—	60	0	12	4	8	3.9	—
19	21	Preoperative	—	—	23	13	18.5	36	21	26	30	15	25	40	23	30	33	1	6	2.5	4.5	—	—
		Postoperative	—	—	—	—	—	—	—	—	40	25	30	—	—	—	40	3	—	—	—	3.1	—
55	33	Preoperative	196	—	40	20	30	—	—	—	90	40	65	—	—	—	90	10	12	0	5	3.4	—
		Postoperative	160	—	19	11	14	25	14	19	35	18	25	50	29	37	40	5	12	4	8	3.05	3.36
56	28	Preoperative	167	—	28	18	24	—	—	—	36	20	30	61	36	50	37	0	9	4	—	—	—
		Postoperative	190	—	—	—	—	—	—	—	—	—	—	—	—	—	43	5	12	2	5	4	—
57	31	Preoperative	101	—	35	20	28	45	20	35	110	40	55	110	45	60	110	0	5	−2	1	2.2	2.2
		Postoperative	—	—	20	10	15	—	—	30	40	15	25	53	15	32	40	5	7	0	2	1.53	1.67

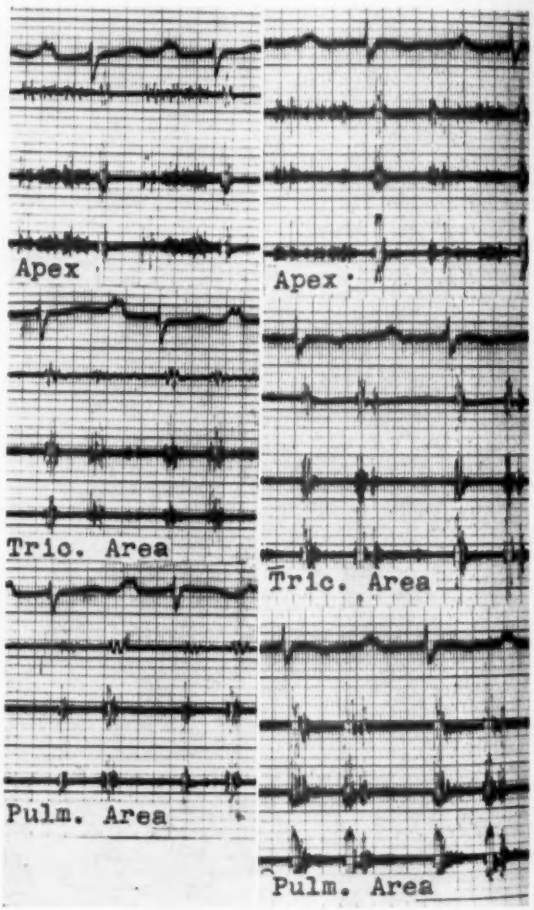
cardiac index have increased in almost all cases. An apparent paradox occurred in one of the patients (Case 47) during catheterization carried out 26 months after the operation. Her clinical condition was excellent, she had had no need of medicines and she had been carrying out her normal housework. The pulmonary pressure before the operation had been 110/46 (mean 80) and was now 40/23 mm Hg (mean 30); the pulmonary capillary pressure had fallen from 40/28 (mean 32) to 25/13 mm Hg (mean 18). The pulmonary capillary pressure after exertion had been 45/35 mm Hg, and now, 26 months afterwards, became 60/35 mm Hg (mean 43). Pulmonary edema appeared immediately. That is to say, in spite of the large fall in pressures after commissurotomy, and the excellent clinical improvement which had enabled this patient to carry out work previously impossible for her, the instability of the pulmonary vascular bed was such that exertion in the laboratory was sufficient to provoke acute pulmonary edema when mean pulmonary capillary pressure reached 43 mm Hg. A biopsy specimen of the lung obtained at the time of operation had shown severe lesions of the internal layers of the arterioles.

Table III lists those cases in which the pulmonary pressure remained the same or increased after the commissurotomy (Group B). In Case 51 in spite of change in pulmonary pressure and postoperative regurgitation the patient showed clinical improvement. In Case 52 there was late rheumatic reactivation following commissurotomy. The third patient (Case 53) showed clinical improvement although he had postoperative regurgitation.

Five cases in Table III (Group C) show a more marked divergence between clinical results and hemodynamic data. In Case 54, the patient's condition deteriorated due to rheumatic reactivation although mean pulmonary pressure fell from 60 to 40 mm Hg and pulmonary capillary pressure from 37 to 20 mm Hg. The second patient (Case 19) showed good clinical improvement although pulmonary pressure gave no indication of this. Results were mediocre in Case 55 in spite of a considerable fall in pulmonary artery and capillary pressures. Case 57 shows how extra-circulatory factors may modify the clinical results. The patient had a slight regurgitation preoperatively and anterior and posterior commissurotomy were carried out without increase in the regurgitation. For a year his condition was very good, then he gained 11 kg in weight and his condition deteriorated. We have observed that the fall in pulmonary artery pressure is not always parallel to the fall in pulmonary capillary pressure.

Correlation of Clinical and Hemodynamic Results: In some cases postoperative catheterization shows a parallelism between clinical results and hemodynamic results; in others there is no correlation. It has been our experience that patients with very high pulmonary artery and capillary pressures generally show good or even excellent clinical results provided they have pure stenosis without complications and that commissurotomy is complete.

We have tried to establish whether or not there is any correlation between clinical results and the data obtained by lung biopsy which we did at every commissurotomy. Almost always the most serious vascular lesions corresponded



A B

FIG. 2. Phonocardiogram. *Left*, before commissurotomy. *Right*, one month after.

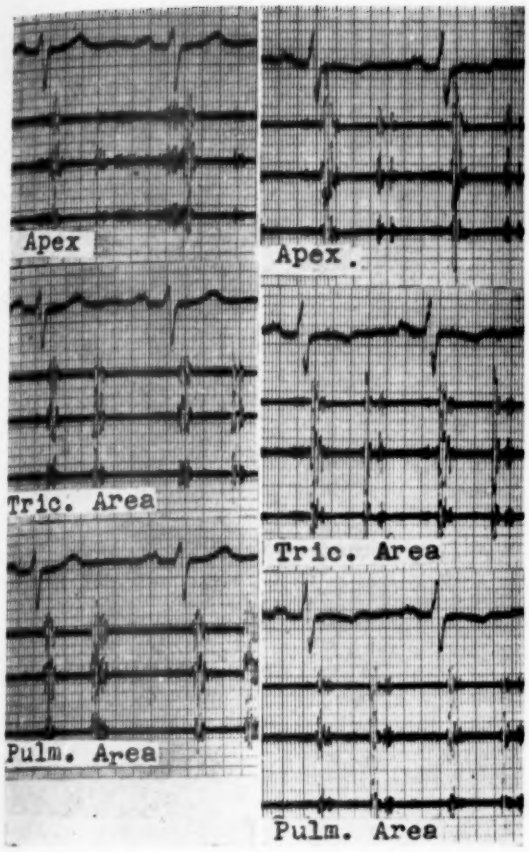


FIG. 3. Phonocardiogram. *Left*, before commissurotomy. *Right*, one year after.

with the highest values of pulmonary artery and pulmonary capillary pressures. Clinical results did not depend on pulmonary vascular alterations. Some patients with very severe lesions of the pulmonary vascular bed showed excellent clinical results. One of the most remarkable cases is Case 44 (Table III). A biopsy showed severe lesions of arteritis, partly necrosing and partly fibrinoid, lesions of periarteritis of rheumatic type, and severe thickening of the intimal layer of the small arteries. Pressures fell considerably and clinical results were excellent. In the second catheterization the mean pulmonary capillary pressure remained unchanged after exertion.

PHONOCARDIOGRAM

In many patients the diastolic murmur disappears during the first few weeks but generally returns. This phenomenon has no physiopathologic significance. When calcification of the valve or other anomalies of the valvular

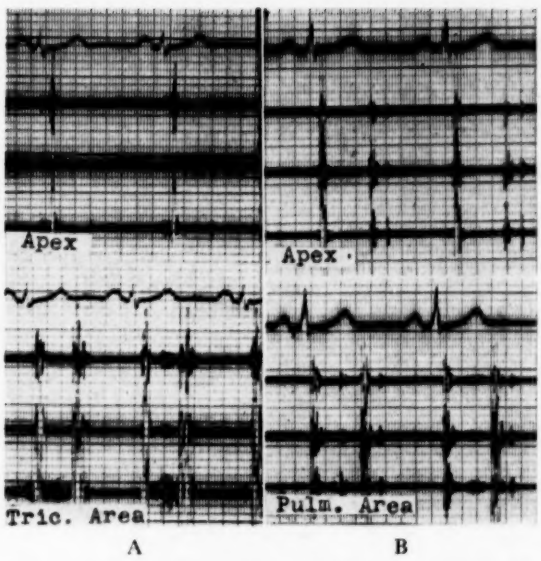


FIG. 4. Phonocardiogram. *Left*, before commissurotomy. *Right*, 13 months after.

system produces systolic regurgitation, the phonocardiogram shows a marked systolic murmur whose intensity varies according to the volume of regurgitation.

The most important signs seen in the phonocardiogram after commissurotomy[14–17] are delay in the opening snap in relation to the second sound (2–OS interval) and shorter duration of the Q-1 interval. These are manifestations of improvement in cardiac activity and were observed in 50 per cent of our cases. We present phonocardiograms of three patients taken at different times in relation to the operation in which these characteristics may be seen (Figs. 2, 3, and 4).

In Figure 2, one month after the operation, the diastolic murmur has decreased considerably, Q-1 interval has decreased from 0.08 to 0.04 second, and 2–OS interval has increased from 0.04 to 0.09 second. The phonocardiogram in Figure 3, eleven months after the operation, shows a decrease in diastolic murmur, and the presystolic crescendo has almost disappeared. Q-1 interval has decreased from 0.10 to 0.07 second and 2–OS increased from 0.04 to 0.09 second. The phonocardiogram in Figure 4 was taken one year after operation. Q-1 interval is 0.10 second before the operation and 0.08 second afterwards, 2–OS is 0.06 second before and 0.12 second afterwards.

ELECTROCARDIOGRAM

Arrhythmias: The electrocardiographic alterations occurring during the course of commis-

surotomy, as described by some authors[18,19,20] and by us,[13,21] such as extrasystoles, paroxysmal tachycardia, and impaired A-V conduction, disappeared completely after the operation. The auricular fibrillation provoked by commissurotomy lasted for only a few hours except in three cases in which it continued permanently. In spite of this the clinical evolution of these three cases was good. Auricular fibrillation appearing after the operation and persisting does not impair the result of the operation. Auricular fibrillation in one patient was resistant to quinidine therapy before the operation, but 18 months after the operation it disappeared after another course of quinidine.

The electrocardiographic alterations resulting from postoperative pericarditis—which occurs in nearly all cases—disappeared completely.

Axis Deviation: Immediately after the operation temporary alterations of the electric axis result from a change in the position of the heart. If these alterations of the electric axis are persistent, they have a different meaning and are the expression of important physiopathologic alterations. Counterclockwise rotation of the axis indicates that the strain on the right ventricle has disappeared because of decrease in pulmonary pressure and that the electrical potentials balance each other (Fig. 5), or that the left ventricle has become enlarged as a result of postoperative regurgitation. In either case, the deviation of the electrical axis to the left has an important physiopathologic significance and is a valuable guide in some cases to the new balance of electrical forces brought about by the operation. The deviation of the axis presented by our patients was of the order of 30 to 40 degrees.

Fifty per cent of our patients showed no permanent postoperative electrocardiographic alterations. In the others we encountered variable electrocardiographic data indicative of the morphologic and physiologic alterations produced by the operation.

P Wave Changes: The alteration of the P wave is one of the most important findings. We have already shown[22] that the earliest electrocardiographic signs of mitral stenosis are in the P wave. This expression of hypertrophy and enlargement of the left atrium may appear before it is revealed by fluoroscopy. A high and double P wave may become completely normal a short time after the operation (Fig. 5). Likewise a high and pointed P wave, showing especially the physiopathologic alterations in the right atrium, may become completely normal (Fig. 6).

Illustrative Cases: The modifications in the orientation of the ventricular potentials, parallel to the hemodynamic alterations are, by themselves alone, the objective demonstration of the efficiency of the surgical intervention. We have chosen four patients with different periods of postoperative evolution which show clearly the result of the hemodynamic

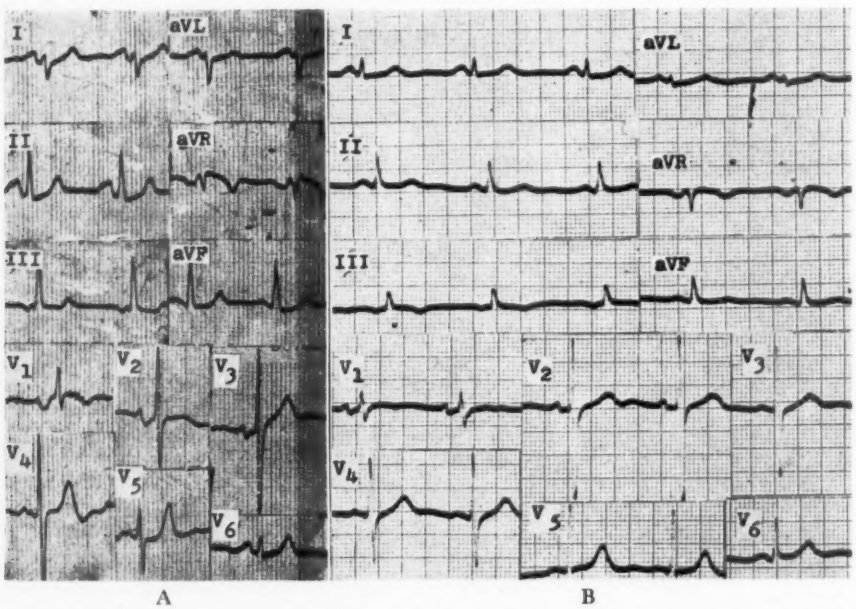


FIG. 5. Electrocardiogram. *Left*, before commissurotomy. *Right*, two and a half months after.

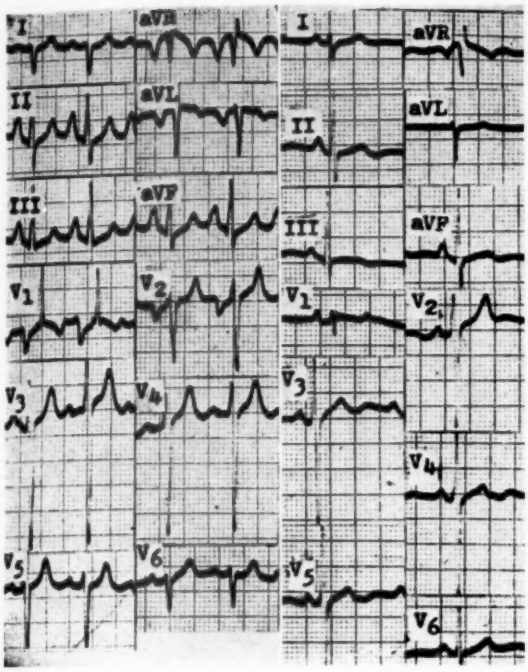


FIG. 6. Electrocardiogram. *Left*, before commissurotomy. *Right*, two months after.

alteration on the electrocardiogram. Figure 5 shows an electrocardiogram, two and a half months after the operation. Deviation of the electrical axis changed from +120 to +60 degrees. Marked alterations have occurred in the morphology of the P wave with decrease in voltage, disappearance of the bifid and chamfered shape, and greatly modified appearance in V_1, V_2 and V_3. A marked decrease is observed in the degree of right ventricular hypertrophy; while S was deep in V_5 preoperatively, it does not appear in V_5 after the operation.

Figure 6 shows an electrocardiogram three months after the operation. No deviation of electrical axis has occurred. The P wave, of high voltage and pointed in II, III, and aVF, and negative and pointed in aVR, V_1, and V_2, becomes almost normal in all leads. The precordial leads show that the signs of right ventricular hypertrophy are very attenuated.

Figure 7 shows an electrocardiogram ten months after the operation, in which the deviation of the electrical axis changed from +120 to +70 degrees. The P wave has become normal, and the signs of the disappearance of right strain and right ventricular hypertrophy are clearly marked. The T wave, deeply negative in V_1 to V_5 before the operation, has become slightly negative in V_1, diphasic in V_2, and positive in the other leads. The pattern of right ventricular hypertrophy has disappeared. The electrocardiogram in Figure 8 was taken 27 months after the operation. The signs of right ventricular strain have disappeared and the pattern of right ventricular hypertrophy is clearly modified. The preoperative electrocardiogram corresponds to a mean pulmonary pressure of 74 mm Hg; the postoperative electrocardiogram corresponds to a mean pulmonary pressure of 35 mm Hg.

In about 40 per cent of the cases the electrocardiogram proved to be a valuable guide in the appreciation of the hemodynamics of the heart after the operation.

VECTORCARDIOGRAM

Before the operation the vectorcardiogram was sometimes useful in differentiating right branch block from right ventricular hypertrophy which was sometimes impossible by the conventional

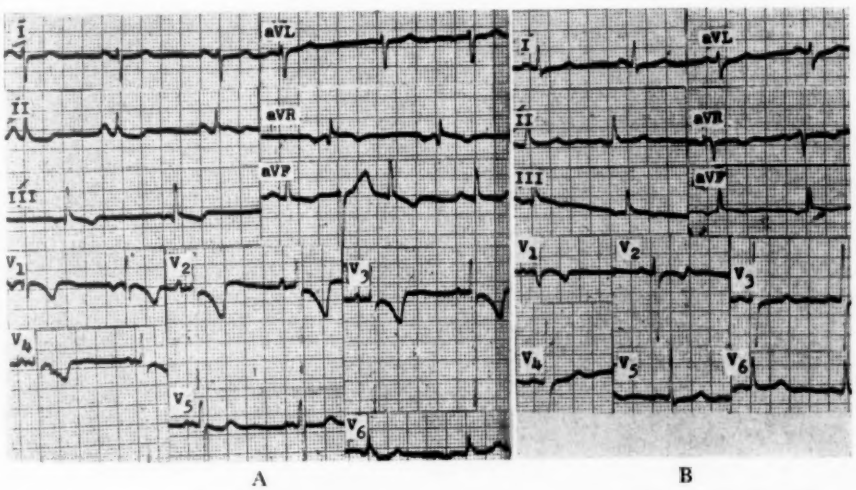


FIG. 7. Electrocardiogram. *Left*, before commissurotomy. *Right*, nine months after.

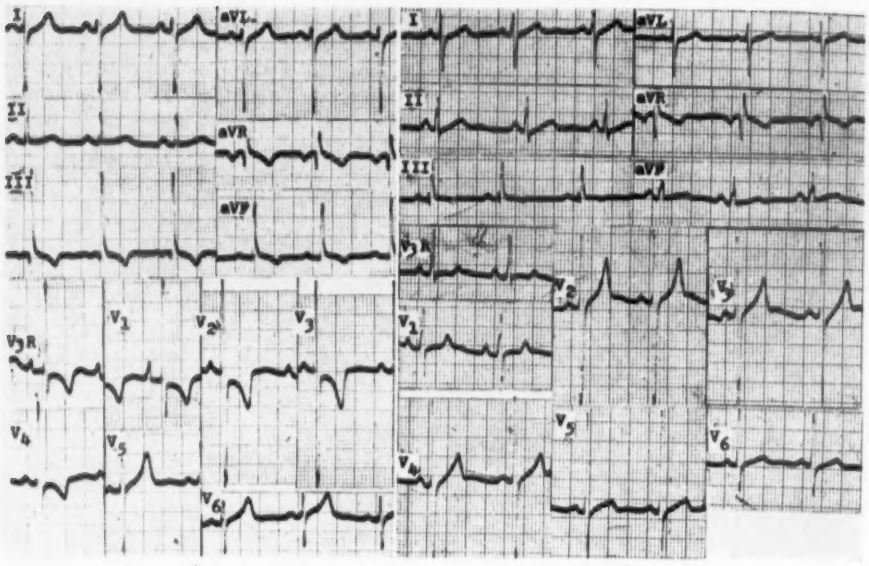


FIG. 8. Electrocardiogram. *Left*, before commissurotomy. *Right*, 26 months after.

electrocardiogram. After commissurotomy in most cases the vectorcardiogram showed no alterations. In the cases in which the conventional electrocardiogram showed disappearance of right ventricular strain and a marked decrease of hypertrophy, the vectorcardiogram showed corresponding marked alterations.

The vectorcardiogram in Figure 9 is from the same patient as in Figure 8. In the frontal plane the loop is developed first to the left, then downward and to the right; in the horizontal plane, also to the front and to the right, in a clockwise direction. Two years after operation the loop is developed in a clockwise direction but much more to the left, and the loop has a morphology similar to a normal vectorcardiogram. In Figure 10 in the horizontal plane the loop, although developed in a counterclockwise direction, is directed entirely to the front. After operation it is developed to the front and to the left, as in the normal pattern.

ELECTROKYMOGRAM

The postoperative modifications in the left atrial pressure pulse are clearly shown by the electrokymogram.[23–25] If the commissurotomy was efficient there were clear signs of normal emptying of the left atrium. In cases of postoperative mitral regurgitation this is clearly indicated by the electrokymogram. This enables us to evaluate the failure of the operation if the regurgitation is intense.

Illustrative Cases: We show two electrokymograms illustrating the two situations. Electrokymograms

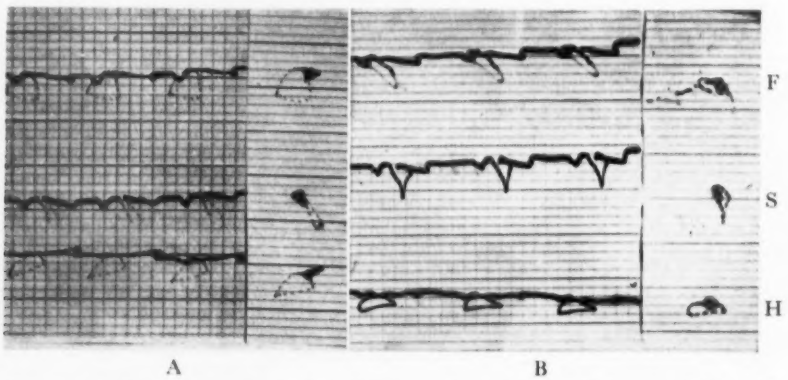


FIG. 9. Vectorcardiogram. *Left*, before commissurotomy. *Right*, one and a half months after.

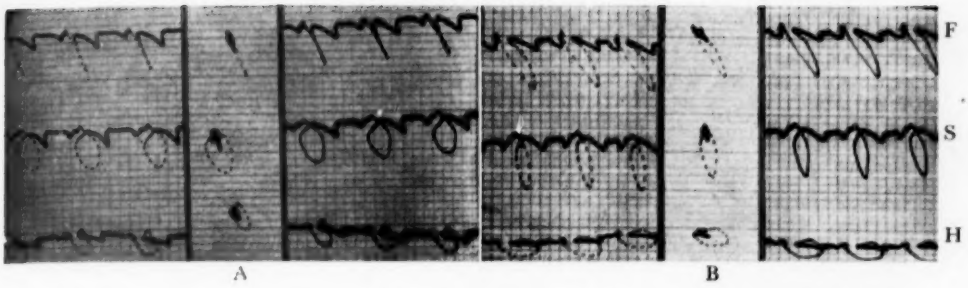


FIG. 10. Vectorcardiogram. *Left*, before commissurotomy. *Right*, two months after.

of the left atrium are shown in Figure 11. In the preoperative tracing, after the opening of the mitral valve which corresponds on the electrocardiogram to the end of the T wave, there is a fall in the tracing indicating difficulty in the filling of the left ventricle (phase of rapid ventricular filling). Immediately afterwards the tracing rises well, showing that the atrium has received sufficient blood from the pulmonary veins and that the pulmonary pressure is high. This is followed by atrial contraction corresponding to the descending part of the P wave and to the PR segment of the electrocardiogram. Only a slight lowering of the tracing is to be seen, showing that the contraction is not efficient. Mitral valve closure corresponding to the S wave on the electrocardiogram occurs with no increase in auricular pressure or volume and this indicates rigidity of the valve. The lowering of the base of the ventricle causes a fall in the tracing (0.08 to 0.12 second after the R wave of the electrocardiogram) and finally the slow filling of the atrium occurs as shown by the rising shape of the tracing. All these characteristics are signs of pure mitral stenosis. The postoperative tracing in Figure 11 was recorded after successful anterior and posterior commissurotomy, with no regurgitation. We note normalization of the tracing. The emptying of the atrium is easy and continuous. After mitral valve closure a normal C wave is formed followed by normal diastolic filling of the atrium.

Electrokymograms of the left atrium are shown in Figure 12. In the preoperative record immediately after the opening of the mitral valve (apex of the V wave of the electrokymogram, corresponding to the end of the T wave of the electrocardiogram) there is a small lowering of the tracing indicating that in the phase of rapid filling of the ventricle the mitral flow is small. Following this the tracing becomes almost horizontal, showing that there has been no change in the volume of the atrium. For 0.24 second a small fall in the tracing is noted, showing the inefficiency of contraction of the atrium. After this contraction and the closing of the mitral valve the tracing rises slightly, showing that, contrary to what normally happens, there has been no increase in the volume of the atrium or increase in atrial pressure. The rapid descent of the tracing which follows corresponds to the lowering of the base of the ventricle. The apex of the C wave of the electrokymogram is 0.08 second after the R of the electrocardiogram. There is no systolic plateau and the atrium fills up to the time of opening of the mitral valve. The electrokymogram shows that there is severe, pure mitral stenosis with valvular rigidity.

At surgery it was found that there was no regurgitation and the surgeon succeeded only in opening the posterior commissure. The postoperative electrokymogram in Figure 12 shows the following: after closure of the mitral valve there is a rapid rise in the tracing and the formation of a small systolic plateau (0.12 second); the C wave has disappeared. The filling of the atrium is so rapid that the variations in pressure and volume resulting from the closing of the

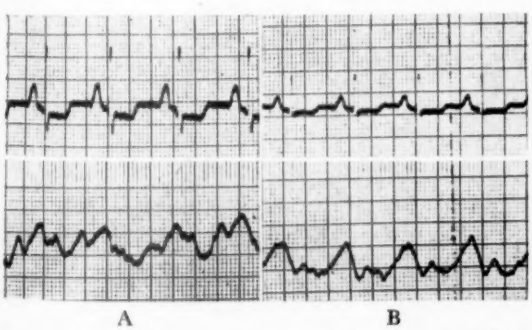


FIG. 11. Electrokymogram. *Left*, before commissurotomy. *Right*, one month after.

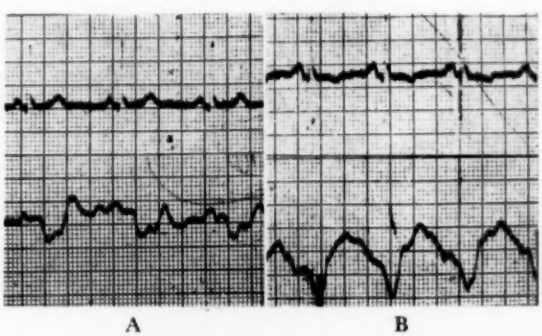


FIG. 12. Electrokymogram. *Left*, before commissurotomy. *Right*, three months after.

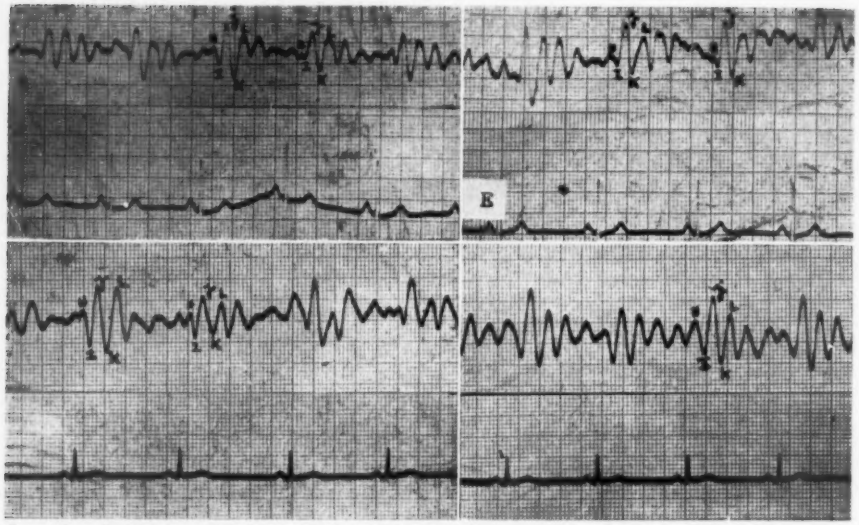


FIG. 13. Ballistocardiogram. *Top*, before commissurotomy. *Left*, after rest. *Right*, after exertion. *Bottom*, two months after.

valve and the lowering of the base of the atrium are cancelled out. The electrokymogram presents a short systolic plateau characteristic of the mitral insufficiency produced by the operation.

BALLISTOCARDIOGRAM

The ballistocardiogram gives information about the alterations of insufficiency of the myocardium and of cardiac contraction, and may help in evaluating the physiopathologic corrections brought about by commissurotomy.

Figure 13 (top) shows a case of mitral stenosis in which the HI segment is very small and does not increase after exertion. After commissurotomy (Fig. 13, bottom) a marked increase in the HI segment is seen both during rest and after exertion. This indicates a clear improvement in the contraction of the myocardium.[25]

RADIOGRAPHY

Although radiology of the chest gives only morphologic information, this is an expression of physiologic alterations.[26,27] In some cases, especially those in which pulmonary artery pressure was very high, postoperative roentgenograms showed a decrease in the bulge of the pulmonary arch, a decrease in the widening of the branches of the pulmonary artery and therefore a decrease in the opacity of the pulmonary hilum, and a tendency to normalization of the pulmonary reticulum. In one-third of the cases the radiologic signs of pulmonary hyper-

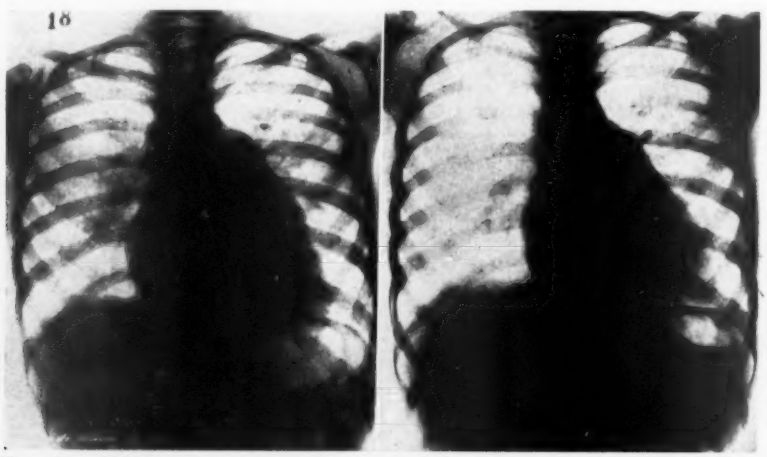


FIG. 14. Radiogram. *Left*, before commissurotomy. *Right*, six and a half months after.

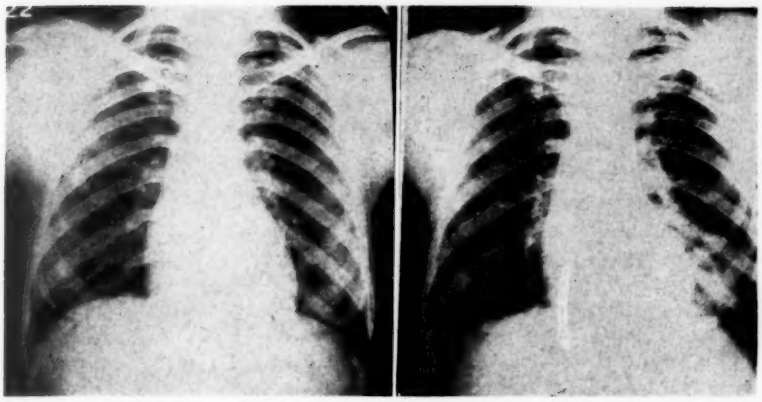


FIG. 15. Radiogram. *Left*, before commissurotomy. *Right*, one year after.

tension disappeared. In the remainder these signs persisted in spite of the disappearance of clinical pulmonary symptoms. In a few cases there was a decrease in cardiac size. When mitral regurgitation developed after commissurotomy, radiography showed an enlarged left ventricle. Reactivation of rheumatic carditis after the operation sometimes provoked cardiac enlargement.

Illustrative Cases: We present roentgenograms of three patients with mitral stenosis at different periods after operation. In each case we note marked decrease of the hilum and pulmonary reticulum.

In Figure 14 the preoperative roentgenogram shows a large bulge of the pulmonary artery due to very high pulmonary artery pressure. Ten months after operation a marked decrease in the bulge on the left contour has occurred.

Figure 15 shows a decrease in diameter and a reduction in the bulge of the pulmonary artery 11 months after surgery. In Figure 16 the roentgenogram taken two years after the operation shows, unlike Figure 15, a normal configuration of the left contour of the heart.

We also observed the disappearance of Kerley's lines.[28] In most patients we did not observe any parallelism between the dimensions of the cardiac shadow and clinical evolution except in those with postoperative regurgitation or insufficiency of the myocardium (those few patients operated on at the begining of this series in whom the myocardial factors were predominant in relation to the mitral barrier). In these cases the increase in cardiac dimensions corresponded to an aggravation of the disease. In other cases the shadow of the heart may increase in size, despite a good clinical result.

PULMONARY ANGIOCARDIOGRAPHY

In some cases we observed a change of the pulmonary circulation in the angiograms. In others, despite the disappearance of the clinical symptoms and a fall of pressure in the pulmonary artery and pulmonary capillaries, selective angiocardiograms maintained the same configurations which they had presented before the operation.

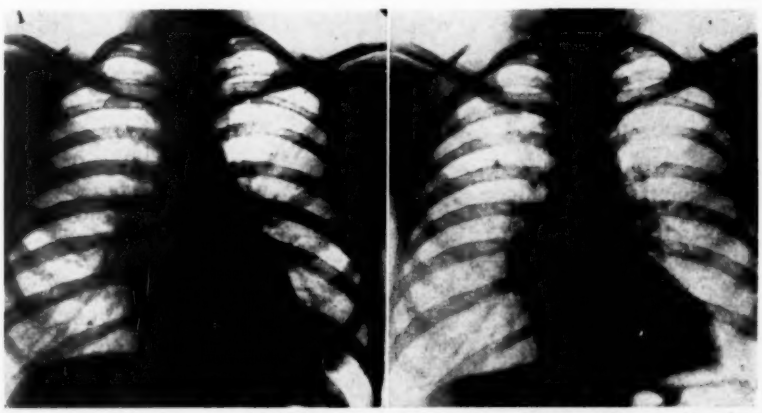


FIG. 16. Radiogram. *Left*, before commissurotomy. *Right*, 28.5 months after.

PULMONARY FUNCTION TESTS

These tests were made one to two months after the operation. We studied mainly the vital capacity, maximum breathing capacity, the coefficient of oxygen utilization, and ventilation. In one-third of the patients we observed an improvement of pulmonary function. Here also we did not encounter any parallelism between clinical improvement and the results of pulmonary function tests.

PSYCHIC ALTERATIONS

Some patients showed slight psychic alterations. We observed more marked psychic disturbances in five patients (2.5 per cent). These later disappeared in three but remained in the other two. Preoperative psychic instability has a fundamental role in the appearance of these disturbances.

Factors Conditioning or Influencing the Physiopathologic Results of Commissurotomy

Mitral Regurgitation: Failure of the operation generally results from marked mitral insufficiency. Postoperative regurgitation, when very marked, aggravates the hemodynamic situation. Of 26 patients with regurgitation, 15 showed considerable improvement because the regurgitation was slight. One of these showed a deterioration immediately after the operation but later improved.

One of the causes leading to postoperative regurgitation is calcification of the valves. We have observed this (with or without ossification) in 7 per cent of the cases. Here the stenosis is due less to fusion of the commissures than to rigidity of the valves.

Arterial Embolism: We have had three cases of death due to postoperative emboli: two of cerebral emboli 24 and 48 hours, respectively, after the operation, and one of multiple emboli of the extremities. Circulation returned to normal in the patient with peripheral emboli. The operation carried out on three patients who had had emboli in the past gave excellent results.

Subvalvular Stenosis: When there is agglutination of the tendinous chordae and the stenosis is subvalvular, this condition is unfavorable to good physiopathologic results.

Myocardial Insufficiency: In insufficiency of the myocardium, revealed by the lack of parallelism between the clinical picture and the intracavitary pressures, which are low,[29] or by marked negativity of the T wave when the first therapeutic doses of digitalis are given,[30] the clinical evolution is the same as if the operation had not been carried out. We observed early aggravation after the operation in two patients with insufficiency of the myocardium.

Insufficient Commissurotomy and Restenosis: Insufficient commissurotomy is another very important factor. Late deterioration may be found following insufficient commissurotomy, of which we had three cases, and following restenosis through rheumatic reactivation, of which we had two cases. If the commissurotomy is complete there is little risk of restenosis. If it is incomplete a second operation will be necessary. Neither severe pulmonary hypertension nor auricular flutter or fibrillation nor pregnancy (three patients were operated on while pregnant) had any influence on the physiopathologic results of commissurotomy.

Clinical Results: In 67 to 70 per cent of our patients the results were either very good or good, sufficient to enable them to return to normal life. The mortality was 6 per cent. Clinical benefits of a subjective order were not always accompanied by corresponding hemodynamic results to justify them.

Finally we should add that in five cases in which it was possible to carry out only a valvular exploration (because of predominant mitral insufficiency) the patients showed subjective improvement during the first two months (psychogenic improvement). They are not included in this series.

Summary

The physiopathologic results of mitral commissurotomy were studied at intervals of several months, one year, two years, and three years after the operation. Pericarditis occurred after the operation in 90 per cent of the cases; and postpericardiotomy syndrome (the so-called postcommissurotomy syndrome) in 12.5 per cent, with no correlation between this and the results of biopsy of the auricular appendage.

The results of mitral commissurotomy were evaluated by preoperative and postoperative cardiac catheterization, phonocardiogram, electrocardiogram, vectorcardiogram, electrokymogram, ballistocardiogram, roentgenogram, pulmonary angiocardiogram, and pulmonary function tests. The physiopathologic data did not always correspond to the clinical results. Sometimes the clinical improvement was greatly superior to the results furnished by these tests.

In this series of 200 patients, excellent or good clinical results were obtained in 67 to 70 per cent of the cases. There was a mortality of 6 per cent.

Finally, some factors are present which condition the physiopathologic results of mitral commissurotomy: postoperative mitral insufficiency, valvular calcification, postoperative emboli, insufficiency of the myocardium, subvalvular stenosis, insufficient commissurotomy, and recurrent rheumatic reactivation, which may cause restenosis.

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Clinical Evaluation of Bilateral Internal Mammary Artery Ligation as Treatment of Coronary Heart Disease*

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THE SURGICAL TREATMENT of coronary artery disease by means of bilateral internal mammary artery ligation is based upon the existence of anastomotic vessels between the tributaries of the internal mammary arteries and the coronary circulation. The existence of such anastomoses and their importance in coronary occlusion have been well documented.[1–12]

Since December, 1954, we have treated surgically 304 patients who have been followed up from six months to four years after the surgical procedure. Like any other attempt to improve myocardial circulation, the ligation of both internal mammary arteries does not influence the arteriosclerotic process of the coronary arteries nor does it improve any previous process of scarring of the myocardium. The foremost indication for this operation is represented by the clinical diagnosis of angina pectoris with or without myocardial infarction.

RESULTS

On this basis, 304 unselected patients, including 207 males and 97 females and ranging from 33 to 83 years of age, were operated upon (Table I). Of these patients, 151 (49.6 per cent) had angina pectoris. This was slight in 4 cases, moderate in 12, and severe in 92, while 43 other patients were suffering from status anginosus. The remaining 153 patients (50.4 per cent) had an episode of myocardial infarction (old in 143 cases and recent in 10 cases), with or without associated angina pectoris (Table II).

TABLE I

Age Distribution of 304 Patients

Age (yr.)	Patients (No.)	Patients (%)
33–39	5	1.6
40–49	32	10.5
50–59	126	41.4
60–69	109	35.8
70–79	30	9.9
80–83	2	0.7

TABLE II

Preoperative Diagnosis in 304 Patients

Diagnosis	Patients (No.)	Patients (%)
Angina pectoris	151	49.6
Slight (1 attack per week)	4	
Moderate (from 1 to 5 attacks per week)	12	
Severe (daily attacks)	92	
Status anginosus	43	
Myocardial infarction (with or without angina)	153	50.4
Old (single)	125	
Old (multiple)	18	
Recent* (single)	10	

* Recent infarction was interpreted as one occurring not more than 30 days before surgery.

In 230 cases (75.6 per cent), the coronary artery disease was complicated by myocardial insufficiency, in 122 (40.1 per cent) by arterial hypertension, in 45 (14.8 per cent) by pulmonary emphysema, in 25 (8.2 per cent) by arteriosclerosis obliterans of the extremities, in 2 (0.6 per cent) by syphilis, and in 1 (0.3 per cent) case by Raynaud's disease.

The evaluation of the clinical results was based on two elements: (1) Clinical improvement based on the sense of well-being, disappearance or reduction of anginal attacks and

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increased tolerance to physical exertion. (2) Electrocardiographic changes following the operation.

The results were further classified as (1) early (within the first 30 days), and (2) late (30 or more days after the surgical procedure) (Table III).

TABLE III
Clinical Results of Surgery

	Early Results*		Late Results†	
	Patients			
	(No.)	(%)	(No.)	(%)
Excellent	31	10.2	27	8.9
Good	143	47.0	135	44.5
Fair	114	37.6	112	37.0
None	15	4.9	17	5.6
Death	1	0.3	12	4.0
Worsening as compared with early result	—	—	14	4 6

* Early results (within 30 days after operation) in 304 patients operated upon by ligation of the internal mammary arteries.

† Late results in 303 patients operated upon by ligation of the internal mammary arteries, in the follow-up period from 3 months to 4 years.

CLINICAL RESULTS

Early Results: Among the 304 cases, 288 patients (94.8 per cent) were improved; of these, the improvement was marked in 10.2 per cent, good in 47.0 per cent and moderate in 37.5 per cent. In 15 cases (4.9 per cent) no appreciable change was noted. A single death occurred on the sixteenth postoperative day due to acute pulmonary edema. A critical review of our 15 failures failed to disclose any reason for it (age, sex, degree of coronary disease, or other associated disease).

All patients underwent the surgical procedure without complications. Routinely, our patients were discharged from the hospital on the seventh postoperative day and returned for a check-up in a month; after that, they were reexamined every three months or, when this was not possible, they were invited to complete a questionnaire.

Late Results: In Table III, it is evident that clinical improvement was maintained after the operation in 274 patients (90.4 per cent); in 14 (4.6 per cent) the improvement was only temporary. Our figures show that, in the follow-up period, the percentage of patients with marked improvement decreased from 10.1 to 8.9 per cent, and from 47.0 to 44.5 per cent, and from 37.5 to 37.0 per cent for the patients with good and moderate improvement, respectively. In addition, the figure for the cases in which no improvement was noted increased from 4.9 to 5.6 per cent.

Fatal Cases: The mortality rate in the postoperative period was 4.0 per cent including 13 patients who died from a minimum of 16 days to a maximum of 13 months after surgery. All these patients were in the advanced age group: two between 50 and 59 years of age, six between 60 and 69, and five between 70 and 79 years of age. In all of them, other complicating diseases were present: cardiac insufficiency in nine, hypertensive heart disease in three, diabetes mellitus in two, and peripheral arteriosclerosis in one.

Nine of these patients had had one or more attacks of myocardial infarction. In one, the infarction was recent; in five, the infarction was anterior; in one, it was posterior; and in 4 others, multiple infarctions were present.

An early good result was noted in five of these fatal cases; a fair result was noted in four cases and no result was noted in 3 cases. One patient died on the sixteenth postoperative day so that the result could not be evaluated. Six patients died in acute pulmonary edema, three of a cerebral vascular accident, two in diabetic coma and two of an acute coronary occlusion. A postmortem examination was performed on the patient who died in the hospital, showing that the immediate cause of death was an acute anteroseptal infarction due to thrombosis of the descending branch of the left coronary artery.

ELECTROCARDIOGRAPHIC CHANGES

After ligation of the internal mammary arteries, electrocardiographic changes were observed in relation to voltage, ST segment, and T waves. Within the first 30 days, the electrocardiogram was improved in 195 cases (64.1 per cent) and a completely normal tracing was obtained in 41 cases (13.1 per cent). In the follow-up period the initial electrocardiographic improvement was not maintained in 10 cases. However, there is no strict relationship between the electrocardiographic changes and the clinical result. In three cases, a normal tracing was not accompanied by disappearance of symptoms; on the other hand, in 11 other cases electrocardiographic evidence of myocardial ischemia was still present even though the patients were symptom-free.

The deep Q waves did not change following the operation, but the electrocardiogram became normal in three cases of left bundle branch block with premature ventricular beats.

From our clinical statistics we had the impression that in spite of the lack of adequate controls, ligation of the internal mammary arteries is more beneficial in cases of anginal pectoris without infarction than in cases following myocardial infarction, and in cases with ischemia of the anterior wall rather than of the posterior wall of the left ventricle. In addition, patients with arterial hypertension seem to benefit most from the operation.

Discussion

The existence of anastomotic vessels between the coronary circulation and the vascular distribution of the internal mammary arteries has been unquestionably shown. On the other hand, the fact that extracardiac anastomoses with the coronary circulation may improve impaired myocardial oxygenation is not a recent concept.[13–17]

All available reports on ligation of the internal mammary arteries seem to confirm the original hypothesis, although demonstration of an increase in myocardial oxygenation after surgery is still lacking. This most probably is only moderate, and doubts were cast as to the actual "revascularization" of the myocardium after ligation of the mammary artery. The clinical improvement has been interpreted as a psychologic effect. It has not been shown as yet that improvement of myocardial ischemia is directly dependent upon the amount of increased blood supply to the myocardium. As a matter of fact those attempts designed to greatly increase this blood supply (e.g., arterialization of the coronary sinus) have not caused greater improvement than other surgical procedures which do not markedly increase the blood supply to the myocardium (e.g., pericardiopexy).

In view of the peculiarities of the coronary circulation, it is safe to suppose that the manner of vascularization and the areas which become vascularized play a more important role than the gross amount of increase in blood supply.

Our laboratory experiments indicate that important hemodynamic changes follow ligation of the internal mammary arteries; these cannot be explained on a psychological basis. Furthermore, the electrocardiographic changes which have been observed in human beings are a proof to the contrary.

Since the first operation performed by Fieschi in 1942,[1] numerous reports have appeared in the medical literature,[2, 18–28] which seem to confirm our experience with the beneficial results of ligation of the internal mammary artery.

Summary

(1) Bilateral ligation of the internal mammary arteries was performed on a total group of 304 unselected patients with various degrees of coronary artery disease. These patients have been followed up from six months to four years after the surgical intervention.

(2) Symptomatic improvement occurred in 288 cases (94.8 per cent); no improvement was noted in 15 cases (4.9 per cent). During the follow-up period, the initial improvement was not maintained in 14 patients (4.6 per cent). No death occurred during or in the immediate postoperative period. Thirteen patients died at various intervals after the surgical procedure; 8 died of cardiac disease, and 5 patients of other causes.

(3) In 195 cases (64.1 per cent) the electrocardiogram improved after surgery, even though no strict relationship was found with the clinical improvement.

(4) Ligation of the internal mammary arteries is a simple surgical procedure which produces an increased blood supply to the myocardium through natural and pre-existing anastomotic channels.

(5) Our results compare favorably with those obtained by more complex and dangerous attempts at myocardial "revascularization."

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Symposium on Phonocardiography (II)

New Method for Analyzing Heart Vibrations

I. Low Frequency Vibrations*

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ALTHOUGH THE TERM "phonocardiogram" was coined by Einthoven who made the first electrical graphic recording of heart sounds in 1894, interest in low frequency heart vibration is fairly recent. Table I was compiled to show the highlights in the history of low frequency recordings.[9,10,12,16–23] As can be seen, most of the authors who have studied low frequencies have believed that in this area of the vibration spectrum, changes occurred which could be correlated with changes in the myocardium. Figure 1, adapted from Butterworth,[3] shows how small a portion of heart vibration energy is audible to the human ear or to the usual phonocardiograph used to record heart sounds, and how much energy is below the audible threshold of 20 cycles. Modern electronic techniques developed for industrial and military uses can now be applied for study of this large area of heart vibration energy, whose character and clinical interpretations are as yet hardly explored.

METHOD OF PROCEDURE

Our procedure for the recording of heart sounds and the analysis of their contents makes use of newly developed equipment. An analyzing system is used which includes a multichannel vibration analyzer. Recent advances in vibration transducers help make possible these more complete studies. Recordings are made from precordial, esophageal, heart surface, and intracardiac pick-ups.

In order to make a permanent recording which is reproducible and which will include all the frequencies contained, we have chosen multichannel FM tape. FM tape is the only magnetic tape recording system which has a frequency response, linearity, and dynamic range consistent with the total energy output of the heart. The use of multichannel equipment and wide instrumentation tape permits us to make many simultaneous recordings of not only intra- and extracardiac sounds but also of electrocardiograms, pulse pressure waves and respirations. It is apparent that comparison of these phenomena in a single heart beat offers great advantage.

Our equipment has been designed for both wide range and selective low frequency analysis recordings. The instruments are able to sense and record for reproduction the very lowest frequencies possible, as well as to cover frequencies well above any that may be encountered. By playback of our original records as a tape loop without transcription we have avoided many of the pitfalls and distortions encountered in transcription. Through the use of a wide range, programmed band-pass filter, any group of frequencies, band-passes and attenuation characteristics can be automatically examined. Special optical and electronic synchronizing and display circuitry complete the automation of these studies. Emphasis of frequencies or areas of wide or narrow band-width can be automatically achieved as indicated or desired. In order to display these complete wide range and versatile

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TABLE I

Development of Analysis of Low Frequency

Year	Investigator	Method	Observations
1937	Eckstein[7]	Studies on muscle strip	Sounds due to muscular contraction
1939	Dietrich and Dunker[4]	Esophageal tube	Slow ventricular vibrations with disease
1940	Kountz, Gilson, and Smith[14]	5 to 250 cps	Large low frequency waves; prolongation of total vibration complex (after myocardial damage)
1941	Kountz and Wright[14]	Man in gas execution chamber	Low frequency subaudible waves chiefly affected in dying heart and myocardial disease
1947	Foulger, Smith, and Fleming[8]	Changes in intensity of vibration after stress	Anoxia lowers frequency of waves
1949	Burger and Koopman[2]	Mathematical analysis	Information on low frequencies of clinical value
1949	Dunn and Rahm[6]	Multiple amplifiers	Importance of low vibrations
1951	Johnston and Overy[13]	Sanborn electromanometer	Velocity curve related to functional status of myocardium
1955	Down[5]	Vibrocardiography (five selective filters separating vibrational energy into distinct frequency bands, lowest 0 to 50 cps)	Believes "principal vibration complex" (30 to 80 cps) of myocardial origin
1956	Groom and Boone[11]	New electronic low frequency transducer	Believe infrasonic frequencies related to mechanical function of heart

analyses, we have chosen a 17-inch cathode ray oscilloscope. This oscilloscope offers an inertia-free, versatile display with uniform frequency response characteristics. Displays of the intensity-modulated qualitative form (as those made by the Bell Laboratories) can be achieved. In addition, a somewhat different, quantitative representation can be readily generated.

In generating this display (see Figs. 1 to 6) the original magnetic tape recording is cut and formed into tape loops. This recording contains the complex heart sounds, electrocardiogram, pulse waves, respiration marks, and phonocardiogram. A tape loop is then marked for optical synchronization at the point of interest. As the tape loop is repeatedly played, it is appropriately amplified, and the complex wave form is examined by a band-pass filter (attenuation outside pass band 24 db/octave, each side). The output of this filter is a sine wave which represents the amplitude of a specific rate of change (frequency) in the complex wave form for each point in time. This sine wave then is the electrical expression of a particular rate of change of the mechanical movement of the precordium. Since the sine wave itself is merely an expression, its wave shape *per se* is not significant. In an effort to see the envelope of the amplitude changes more readily, full wave rectification of the sine wave is employed. It is this display which is presented in Figures 4 through 6. Our most recent work includes integration after rectification so as to make a more smooth wave, and subsequent modulation with a higher frequency. Through this approach a smooth line record is achieved which is still readily measurable from its own baseline. This approach appears to be ideal for the more localized studies

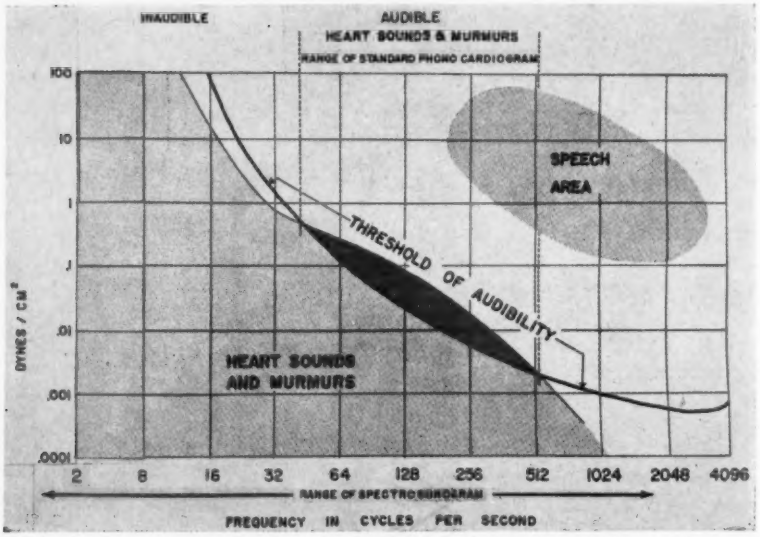

FIG. 1. This graph indicates an average threshold of audibility and shows how small a portion of the vibratory spectrum of the heart lies above the threshold of audibility.

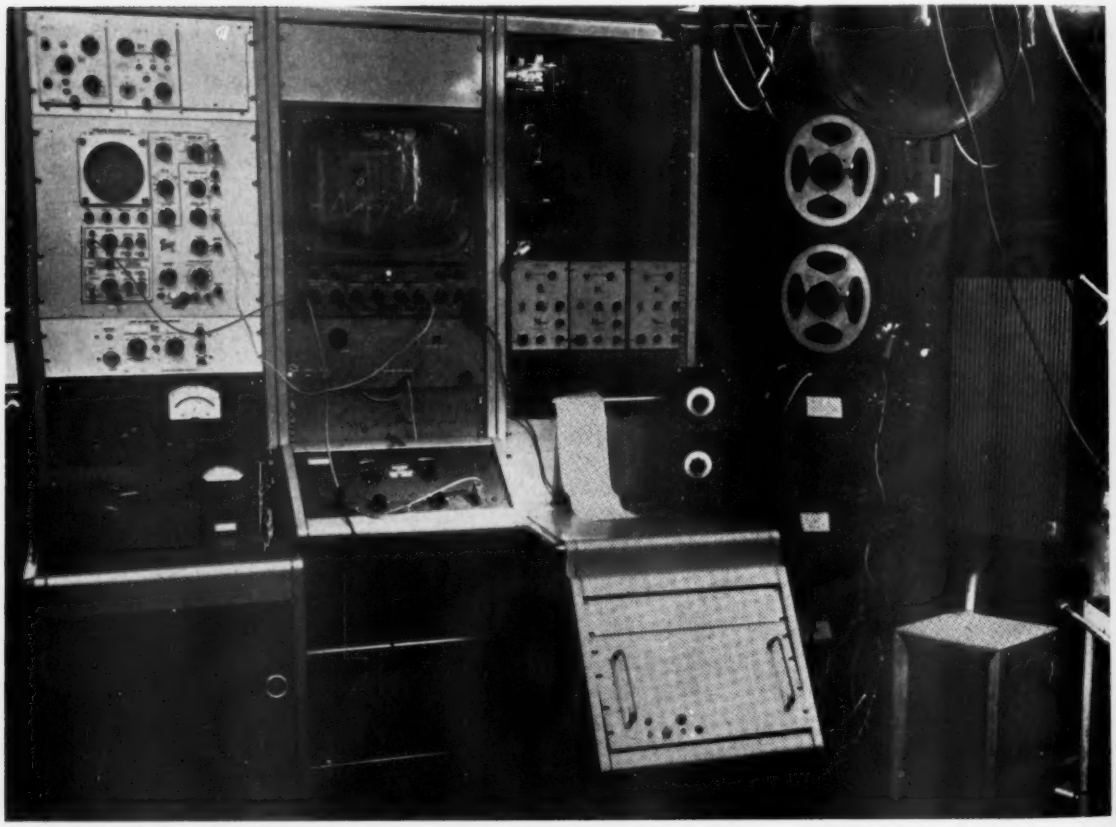

FIG. 2. This photograph demonstrates our present recording, playback, and analyzing instrumentation which physically and functionally acts as one single unit.

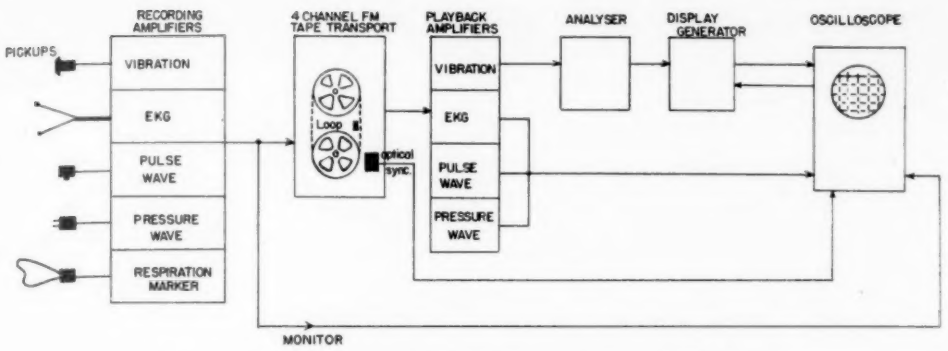

FIG. 3. A block diagram of the equipment pictured in Figure 2.

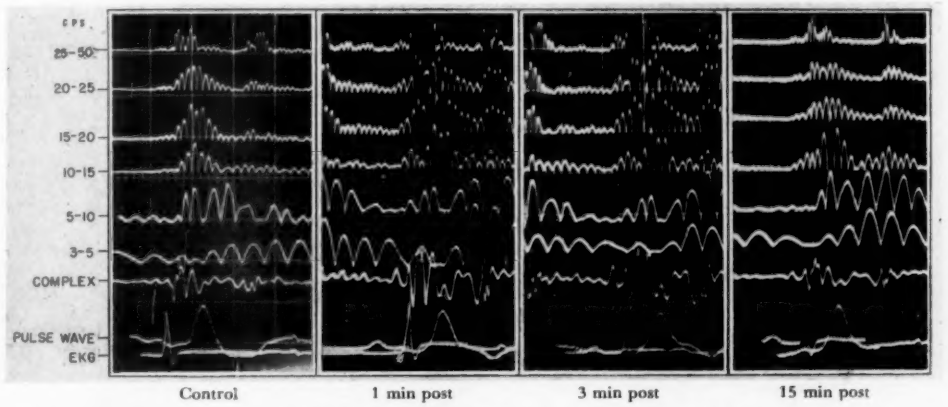

FIG. 4. Effect of smoking. A 20-year-old male in supine position. Altec microphone at apex. Control record and records taken one, three, and fifteen minutes after smoking one cigarette in ten minutes.

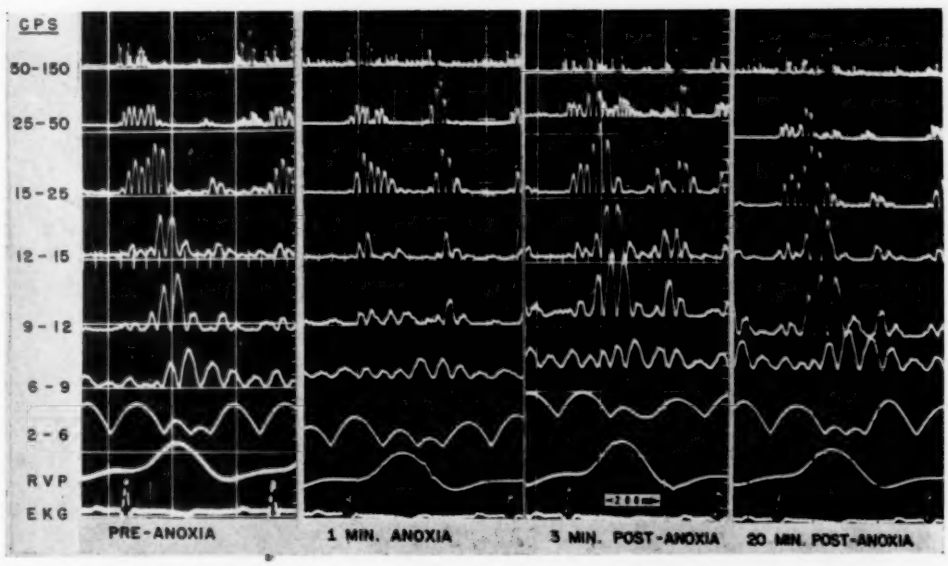

FIG. 5. Effect of anoxia. Right ventricular intracardiac sound recording with Gulton microphone. Records taken on a dog under anesthesia; before, during one minute of total anoxia, and three and twenty minutes after anoxia.

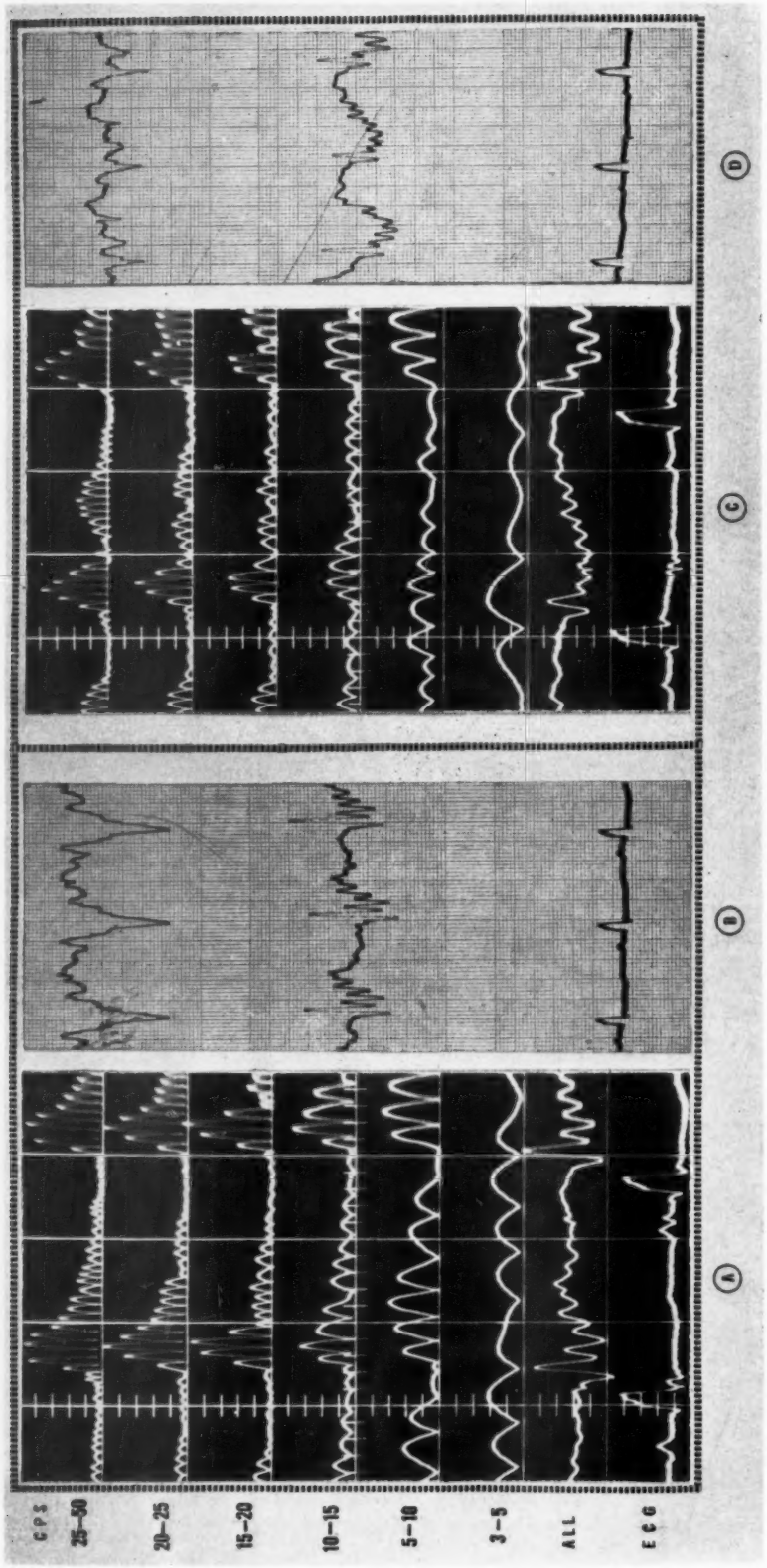

FIG. 6. (A) and (C) are frequency-analyzed surface tracings of a dog before and after coronary embolization. (B) and (D) are complex intracardiac (top) and surface recordings (center) taken before and after sphere embolization. Lead 2 electrocardiogram is seen at the bottom of all tracings.

in which critical frequencies are to be extracted and measured. By recording these images photographically with fine large format equipment a faithful permanent record is produced.

Our working procedure with human subjects includes complete electrocardiograms taken at control points with lead 2 used as a continuous timing reference; respiratory and pulse waves are also recorded. Often a standard phonocardiogram is taken simultaneously. Patients are placed in a supine position on a horizontal foam rubber table. Vibration pick-up is most often placed at the apex. Normal control records are made with the patient in a horizontal resting position and breathing normally. Stress records can then be made directly, either by tilting the patient or by changing the respiratory gases. Tape recordings are made which include our own running commentary, the history, immediate observations, and conditions. After recording, the patient is free, and we are able to complete our selection of the records and the analysis at a later time.

In animals, both precordial and intracardiac recordings are taken. Electrocardiograms, for comparison and timing, and respiration and blood pressure curves are recorded as indicated. The ability to make continuous recording during a lengthy procedure and then to select specific areas for analysis is found convenient.

Illustrative Recordings

The present physical form of the apparatus now being used is shown in Figure 2; Figure 3 is a block diagram of this equipment.

Figure 4 shows the effect of smoking on a normal male at rest. Note the different characteristics of the wave forms at the band frequencies chosen, the marked changes in these sounds produced by the cardiovascular responses to smoking, the repetition of wave forms after alteration by smoking, and the return to a pattern in all frequencies, easily recognizable as the control tracing.

Figure 5 illustrates changes induced by anoxia. Note that the electrocardiogram showed no definite changes. This was true in this and other instances even when the anoxia was carried to the point of ventricular fibrillation. On the other hand, the spectrosonogram changed materially, often in as soon as eight seconds. Also note the return of the records to the preanoxic pattern.

Figure 6 is an example of records taken before and after the injection of plastic microspheres into the coronary arteries.[1] Here again, while autopsy showed many 1 to 2 mm infarcts in the heart muscle, electrocardiograms taken within a half hour of onset of these infarcts showed no change. The vibration analyses were materially altered. Note also that there were distinct changes in the complex intracardiac and surface recorded tracings, yet the difficulty of analyzing these changes without the relative simplicity afforded by frequency analysis is clear.

Conclusions

Figures 1 through 6 indicate that there are striking changes in some, or usually all, the wave bands selected. At the 20 to 25 and 25 to 50 cps level the records are dominated by the valve sounds. At the lower frequencies, the influence of valve sounds diminishes or nearly disappears, leaving sound produced by movement of other structures or elements of the heart and blood. It is too early to attempt an interpretation or correlation with cardiac function. However, it is certainly apparent that the records clearly show changes which further study should prove rewarding in terms of affording a new approach to the use of heart sounds for diagnosis and evaluation of cardiac function.

Summary

(1) A brief review of the history of low frequency heart sound recording is outlined.

(2) A new method of heart sound analysis for all frequencies is presented but with emphasis on low frequency components.

(3) Illustrations are given indicating the use which may be made of this technique in future study of low frequency sound.

Acknowledgment

We are very much indebted to the generosity of Mr. Morris Press and Mr. Clarence Meyers.

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The "Displacement" Vibrocardiogram of the Precordium in the Low Frequency Range*

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A large band of precordial vibrations originating from the heart and large vessels is below the auditory level (infrasonic and subliminal bands according to Zalter and Luisada[1]). Much work has been done in the past hundred years to investigate precordial movements (mechanical cardiography, "linear" cardiography, kinetocardiography, thoracic ballistocardiography). Different recording techniques and questionable interpretation, however, have cast doubts as to the validity of mechanocardiographic findings. I have developed a method for recording the tracing of *acceleration* of the precordial pulsations. The present study will be devoted to the investigation of precordial *displacement* tracings. Standard graphic patterns, which had been described in previous reports, have been verified by this method, which has also permitted the collection of new data concerning the physiological meaning of the tracings.

Method

The theoretic value of graphic tracings related to cardiovascular activity depends upon: (1) correct physical properties of the recording instrument; (2) an understanding of physical and physiological facts expressed by the tracing; and (3) avoidance of possible artefacts caused by extracardiac and extravascular factors. Our experiments and their interpretation have been based upon an attempt to meet these requirements.

A pressure operated microphone† utilizing the condenser (or capacity) variation principle has been used to pick up the precordial vibrations. In this microphone, the displacement of the diaphragm is proportional to the change in pressure. The amplifier-galvanometer-filter system used in this study has a linear response.‡ The electronic band-pass filter§ offers independently adjustable high and low cut-off frequencies from 0.02 to 2000 cps with an attenuation rate of 24 db/octave. The distortion introduced by the filter does not exceed 0.5 per cent of the output signal.

The microphone was held in place by a suction cup and exerted only minimal pressure on account of its weight of 75 Gm. Serial records have been taken by using different bands between 3 and 50 cps, simultaneously with the electrocardiogram or phonocardiogram, the direct body velocity ballistocardiogram (method of Arbeit-Linder), and, in a few cases, intrathoracic pressure curves from cardiac patients submitted to cardiac catheterization.

Two hundred and fifty precordial vibrocardiograms were taken in 10 young subjects aged 5 to 21 years. The subjects were in the supine position. Different locations of the pick-up on the chest wall were tested scanning the entire precordium. Variations due to respiratory influences have been studied during normal respiration, inspiratory and expiratory apnea, and forced respiration.

Additionally, an electromagnetic, two electrodynamic, and one crystal microphone with "linear" response have been tested by using the same frequency band of the filter. Data concerning time values have been based on the measurement of 15 consecutive cycles.

Results

The Precordial "Displacement" Vibrocardiogram: A typical and reproducible pattern of the vibrocardiogram was found in the 5 to 25 cps frequency range during normal respiration, if the tracing was recorded in the fifth intercostal space 1 cm from the left sternal border (Fig. 1) or over the xiphoid. No significant difference has been found between tracings re-

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† Manufactured by The Altec Lansing Corp., Beverly Hills, California.

‡ This system was described by Zalter and Luisada.[1]

§ This was a Krohn-Hite band-pass filter No. 330-A, manufactured by the Krohn-Hite Company, Cambridge, Massachusetts.

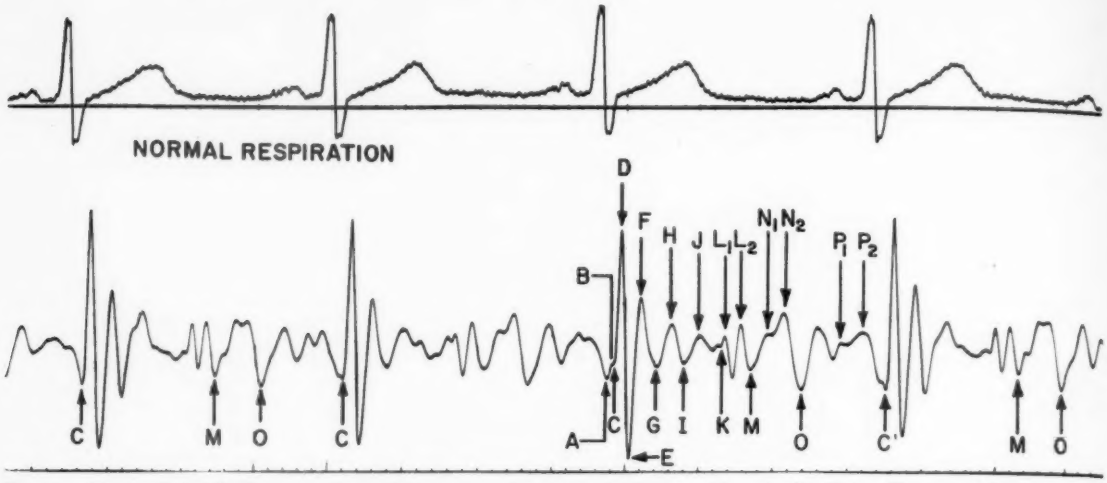

FIG. 1. Precordial "displacement" mechanogram of a normal 17-year-old male (J. K.). Tracing taken from the lower end of the sternum during *normal respiration*. Repetitive respiratory variations of C-G-H and M-N-O. Vertical time lines mark distances of 40 milliseconds.

corded over these two locations on the chest wall in normal subjects. The oscillatory pattern is quite similar to that previously described for precordial acceleration.[2]

Forced inspiration, inspiratory or expiratory apnea, different kinds of filters, or other types of microphones, as well as different locations on the chest wall, modified the standard oscillatory pattern (Figs. 2 to 5).

Some segments of the precordial tracing may be compared with those of ultra-low frequency ballistocardiograms.[3,4] The precordial tracing in Figure 6 has been recorded with reversed polarity in order to emphasize this resemblance.

Following the previously described method, each peak of the tracing has been labeled with a letter in alphabetical sequence from A to P (Fig. 1). Thus, each segment was labeled with three letters. The first letter indicates the upstroke; the second, the peak; and the third, the end of the segment.

Description of the Vibrocardiogram: The description, time relationships, and tentative in-

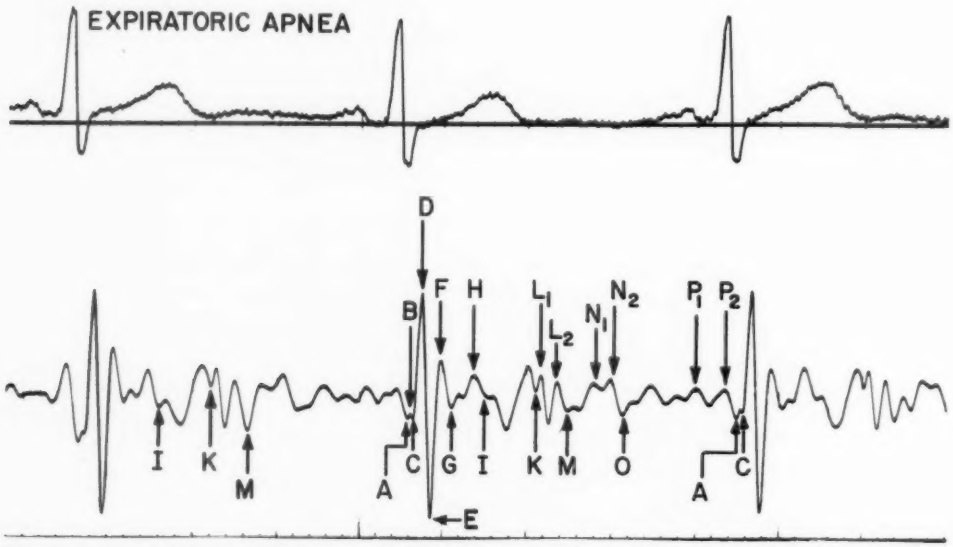

FIG. 2. Same subject as in Figure 1. Tracing taken in *expiratory apnea*. Consistent form of A-B-C and I-J-K. The ascending branch of G-H is consistently double-peaked. M-N-O has a smaller amplitude than during normal breathing.

TABLE I
Description and Interpretation of Segments of Vibrocardiogram

Segment	Description	Average duration (milliseconds)	Time coincidence	Tentative interpretation
		Systole		
Q(ECG)-A		0–50	Spread of ventricular excitation; partial myocardial contractions without measurable rise of intraventricular pressure; electropressor latent period (Schuetz)	
A-B-C	A small, rapid vibration, often superimposed on the C-D upstroke; may be inverted or absent; when absent, it appears with the patient in apnea	20–40	A: R-S complex of electrocardiogram; first slow vibration of 1st heart sound; slow rise of intraventricular pressure. G peak of velocity ballistocardiogram	First part of ventricular tension period (1, left ventricle; 2, right ventricle)
C-D-E	Slender, high, peaked vibration	40–60	C: Junction point of S and S-T of electrocardiogram; first large vibration of 1st heart sound; middle of G-H upstroke of velocity ballistocardiogram	Second part of ventricular tension period (left ventricle) C: Onset of right ventricular ejection
E-F-G	Slender, high peaked vibration	60–80	E: Carotid upstroke; upstroke of "c" wave of jugular phlebogram; H peak of ballistocardiogram	E: Onset of left ventricular ejection
G-H-I	See respiratory changes	60–80	G: Junction point of S-T and T (steep rise of T) of electrocardiogram; carotid shoulder;[5] systolic peak of phlebogram	
I-J-K	Mostly biphasic, convex or concave wave, formed by two vibrations; see respiratory changes	40–120	I: Peak of T wave of electrocardiogram K: End of T wave of electrocardiogram; first vibration of 2nd heart sound; L_1 peak of ultra low frequency ballistocardiogram	Late ventricular ejection; rebound of blood from the periphery into the great thoracic vessels
		Diastole		
K-L-M	Two peaked oscillations	80	2nd vibration of 2nd heart sound	Closure of and impact of blood on the semilunar valves
M-N-O	Configuration depends upon respiration; see respiratory changes	150	M: Opening of atrioventricular valves N: 3rd heart sound; first diastolic peak of right ventricular pressure curve	Rapid ventricular inflow
O-P	May be absent	Inconstant		Diastasis (Henderson)
P-A	Three waves; see respiratory changes	120–200	Approximately P-Q interval of electrocardiogram; 4th heart sound	Atrial phase; the waves probably correspond respectively to the atrial contraction (dynamic phase of Wiggers) and outflow

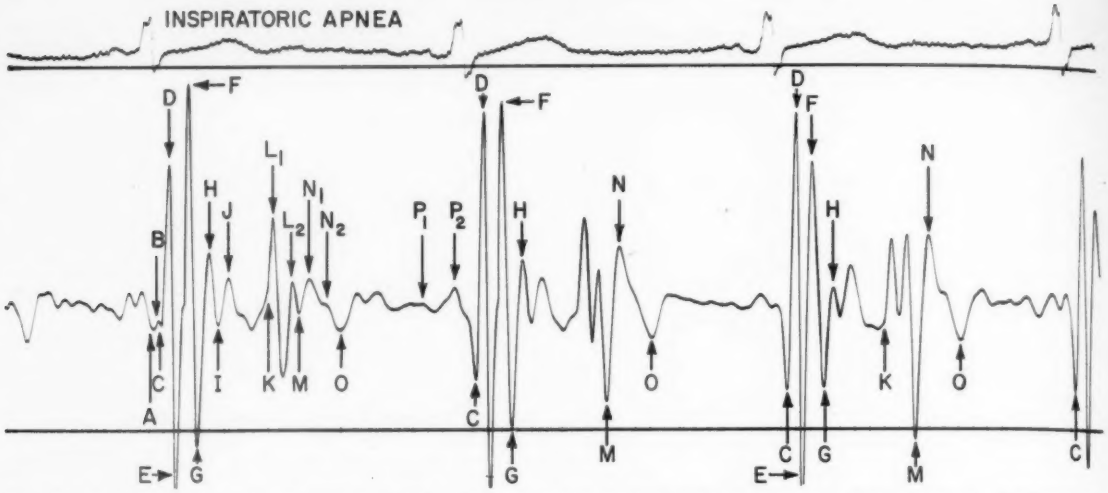

FIG. 3. Same subject as in Figures 1 and 2. Tracing taken during *inspiratory apnea*. Note the gradual decrease in amplitude of F and G-H on held inspiration. Progressive increase in depth of C and M. Gradually, D becomes the highest oscillation of the tracing. Also N becomes higher as inspiration is held on. Flatter baseline in the second part of diastole in comparison to Figure 1.

TABLE II

Description of Respiratory Variations in Vibrocardiogram

Segment	Normal respiration	Expiratory apnea	Inspiratory apnea
A-B-C	Inconstant (Fig. 1)	Constant (Fig. 2)	Becomes progressively deeper (Fig. 3)
C-D-E	Constant in form	Constant	Significant, transient increase in amplitude
E-F-G	Constant in form	Constant, small	Initially, the largest deflection of the tracing; later on, it becomes progressively smaller
G-H-I	Single or double peaked, depending upon the respiratory phase	Constant, small, double peaked	Slender, high, peaked, progressively decreasing in amplitude
I-J-K	Very inconstant	Constant, two oscillations, the second of which is higher	Very constant: the first oscillation is higher
K-L-M	Constant	Constant	Initially: L_1 very high, L_2 small; on held inspiration: L_1 decreases in amplitude, M becomes very deep
M-N-O	Slightly inconstant, high	Small	Very constant, very high
O-P	Constant, well pronounced; its presence depends on the length of the period; it may be absent	Small, inconstant	Small, almost isoelectric
P-A	Slightly variable, made of two or three oscillations	Small	Small

terpretation of the oscillations are presented in Table I.

Respiratory Variations: The respiratory variations are presented in Table II.

Influence of the Apparatus: Different kinds of filters (Fig. 4) or microphones (Fig. 5) may influence the standard oscillatory pattern. The frequency range 5 to 25 cps contains comparatively few of those high frequency components which coincide with valvular events. (See in Figure 4 the absence of A-B-C and the small K-L-M waves.)

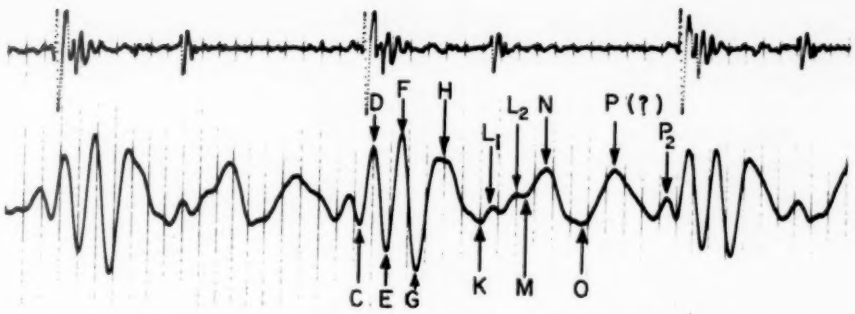

FIG. 4. Same subject as in Figures 1 to 3. Tracing taken during normal respiration. *Upper tracing:* phonocardiogram 100 to 250 cps. *Lower tracing:* precordial vibrocardiogram 4 to 20 cps. Capacitance microphone like in the preceding tracings. Absence of A-B-C in this fre uency range. No I-J-K segment. Low L_1 and L_2 waves. P (?) is apparently an artefact.

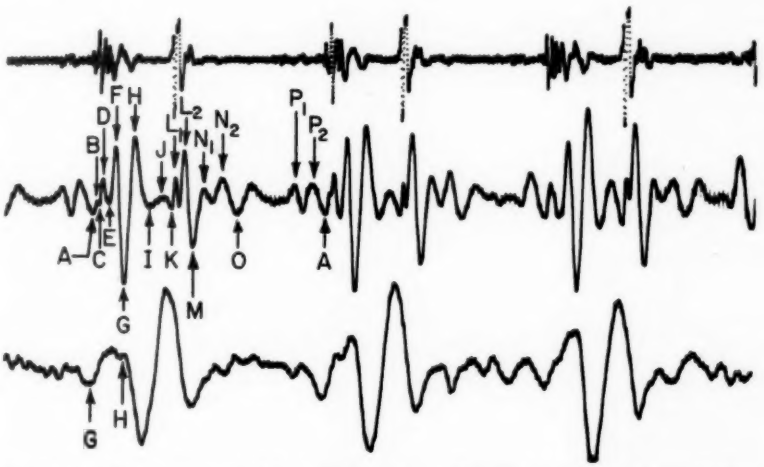

FIG. 5. I. H., a 21-year-old, normal male. *Uppermost tracing:* phonocardiogram 150 to 250 cps. *Middle tracing:* precordial vibrocardiogram 5 to 25 cps. Crystal microphone. *Lower tracing:* velocity ballistocardiogram (method of Arbeit-Linder). Vertical lines: 20 milliseconds. Low C-D-E deflections due to the different frequency response of the receiver.

The pattern of the precordial tracing recorded through a crystal microphone (Fig. 5) slightly deviates from the standard pattern. The A-B-C and C-D-E segments are slightly distorted.

Low Frequency Intracardiac Tracings: Intracardiac atrial tracings (Fig. 6) in the low frequency range (5 to 25 cps) present a pattern which is similar to that of precordial vibrations. In Figure 6, the left atrial pressure tracing appears as a mirror-like image of the precordial tracing. Systematic studies in animals with intracardiac and precordial low frequency tracings are in progress.

DISCUSSION

SYSTOLE

No known graphic method permits the *exact* determination of the initiation of ventricular systole. In phonocardiography, the time interval between the Q wave of the electrocardiogram and the first heart sound varies with the frequency range (Maass and Weber,[7] Trendelenburg[8]). Intracardiac pressure tracings are also modified by the mechanical characteristics of the system. No exact method indicating the beginning of the tension period is universally accepted. Consequently, attempts to label individual oscillations of the vibrocardiogram on the basis of time coincidences with other cardiovascular tracings should be considered as rough estimations whenever they try to evaluate periods in milliseconds. Also, phase shifts due to oscillatory properties of different mechanical systems, like the body or its parts,

probably limit the accuracy of time measurments to about 94 per cent.[9] Certain definitions, like "electropressor latency period"[10] are of high theoretic value in evaluating the efficiency of mechanical recording techniques and provide useful help in the understanding of controversial data. The present findings correspond to data published on chest wall accelerograms;[2] however, it is appropriate to consider the Q-A interval as a time interval depending upon vibratory conditions.

The time interval between the Q wave of the electrocardiogram and the first large vibration of the first heart sound has been considered by Luisada[11] as being due to myocardial tension. Holldack[12] also measured the "transformation time" from the Q wave of the electrocardiogram to the first large vibration of the first heart sound. The precordial vibrocardiogram permits division of this period into two parts: (1) electropressor latency period (*Q-A interval*); and (2) ventricular transformation period or first part of left ventricular tension (*A-B-C segment*). (The right ventricular contraction starts slightly later.[11,17]) Any prolongation of one or both of these time intervals (described in previous reports as "delay of the first sound"[13] or "electroacoustic latency period"[14]) should be investigated as to how far one or the other component is responsible for the prolongation. This time interval plays an important role in physiology.[15] In pathologic states, it has been found prolonged in patients with mitral stenosis[13] and arterial hypertension.[16] The ballistocardiogram has also been found distorted in these conditions because pathologic waves occurred during the same period.[5]

The *C-D-E segment* probably corresponds to the second part of left ventricular tension[17] (pressure rise period of Holldack), and includes the right ventricular tension period. Braunwald, Fishman, and Cournand[18] found an interval of 0.115 second between the onset of the Q wave of the electrocardiogram and the onset of left ventricular ejection. This time interval equals the Q-E interval which includes the electropressor latent period and isometric tension time (Q-A plus A-C plus C-E = 80 to 110 milliseconds). The assumption, that the peak E of the precordial tracing coincides with the peak H_L of the ballistocardiogram, is supported by the data of Reeves and coworkers, who

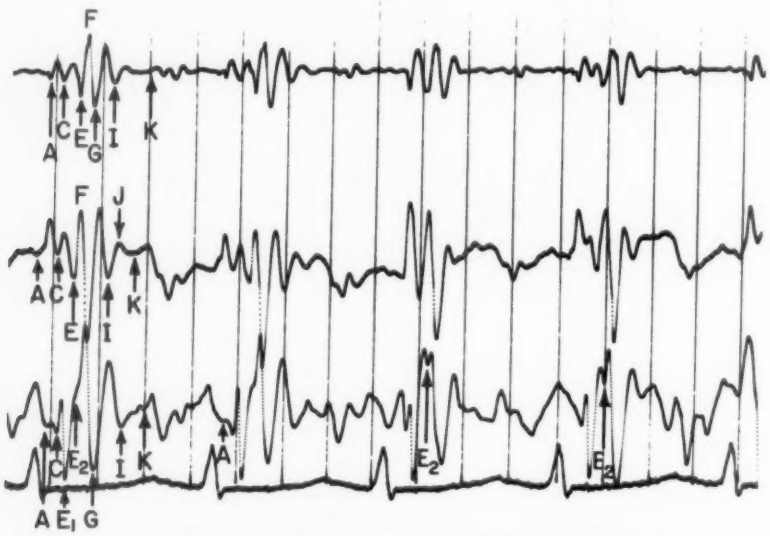

FIG. 6. J. S., a 27-year-old female with mitral stenosis. From above: (1) Intracardiac (left atrial) phonocardiogram 25 to 50 cps. (2) Left atrial pressure tracing (P23D). (3) Precordial vibrocardiogram 5 to 25 cps. Crystal microphone. (4) Electrocardiogram (lead 2). Vertical lines every 200 milliseconds. Early start of A on atrial pressure tracing. The precordial tracing shows a delay of G and I, probably due to delayed filling of the pulmonary artery. The E-F segment is double-peaked, marking the superimposed impulses of left and right semilunar opening. E_1 probably coincides with the opening of the aortic valve. E_2 coincides with E of the pressure tracing.

measured 0.116 second for the Q-H_L interval.[5]

DIASTOLE

The individual oscillations of this period are less consistent than those of the systolic phase. Distinct oscillations in diastole appear during normal respiration over the xiphoid process (Figs. 1, 4 and 5).

The vibrocardiogram seems to hold promise for a more exact differentiation between the two components of the second heart sound (L_1 and L_2) and for the graphic distinction of the rapid ventricular inflow (M-O). Although more observations are needed to confirm previous data obtained with precordial tracings simultaneously recorded with intraventricular pressure curves,[2,19] it seems probable that K indicates the time of semilunar closure, M that of atrioventricular opening, and N that of the third heart sound.

RESPIRATORY VARIATIONS

The respiratory variations presented in Table II and in Figures 2 and 3 reveal that the diastolic oscillations of the vibrocardiogram are not caused by passive vibrations and resonant artefacts.

The constant pattern of the wave A-B-C in expiratory apnea supports the concept that the initiation of the rise in intraventricular pressure may be graphically distorted in tracings of the chest wall by rotatory heart movements and respiratory displacement of the heart.

The progressive increase in amplitude of C-D-E and E-F-G in inspiratory apnea probably corresponds to the initial increase of right ventricular output.

A general increase in amplitude of ballistic waves due to inspiration has repeatedly been described. The precordial vibrocardiogram permits a detailed analysis of respiratory changes. Figure 3 shows a significant decrease in amplitude of H in inspiratory apnea accompanied by a progressive deepening of M. A complete understanding of these variations requires further observations made with simultaneous pressure records. However, both systolic and diastolic respiratory changes of the tracing indicate a close connection with hemodynamic and not with body resonance factors.

PHYSICAL INTERPRETATION

The question whether or not pressure changes of the air column which is between the precordium and the diaphragm of the microphone modify the displacement record of the thoracic wall needs basic clarification. Experiments dealing with this problem have been started. It is likely that damping and resonance factors due to the properties of the chest wall and of the air contained in the bell may cause some degree of distortion.

The vibrations under discussion belong to the infrasonic band of the phonocardiographic spectrum.[1] Such "sound" tracings have been published by Johnston,[20] Rosa,[21] Kountz and coworkers,[22] and Dunn and Rahm.[23] Most of the direct thoracic kinetocardiograms and acceleration ballistocardiograms[2,24–28] present a similar pattern. This similarity reveals the existence of a close relationship between the precordial vibrations and those recorded by ultra-low frequency ballistocardiograms. There is no doubt that the precordial vibrations are originated by cardiovascular forces, part of which are also revealed by the body acceleration ballistocardiogram. The remarkable constancy of the precordial patterns seems to prove that the distorting effect of artefacts inherent in this method is less significant than in indirect ballistic methods.

PHYSIOLOGIC INTERPRETATION

An exact evaluation of cardiovascular forces is at present impossible. All current ballistocardiographic techniques are subject to significant limitations and distortions.[3,29] Factors like damping or resonance effects exerted by connective or other soft tissues, limbs, body weight, and position and rotation of the heart probably affect and modify all types of mechanocardiographic tracings. Starr[30] emphasizes the diagnostic value of ballistocardiographic tracings in systole while, as he points out, aftervibrations occurring in diastole decrease the accuracy of the tracings. Talbot and Harrison[29] conclude that relative motions of various parts of the body distort all ballistocardiograms through summation, complex mass, and loading effects. Passive limb masses further distort total body vibrations.[31] Frederick and Eddleman[6] state that no single segment of the ballistocardiogram is due to a single force, and this is probably true also for precordial vibrations. Graphic evidence for the contribution of both the left and right side of the heart to the precordial tracing may be seen in Figure 6, in which time, amplitude, and morphologic differences (double-peaked E) between intracardiac and precordial tracings clearly dis-

close the summation of complex pressure components.

The upper limit of the calculated linear frequency response (30 to 40 cps)[5] of the ultra-low frequency ballistocardiographic systems varies with the square root of the weight of the subject. The frequency limit (25 cps) used in our study may decrease the possibility of distortion due to individual differences in body weight. The elimination of the role played by the limb masses further represents an advantage by reducing artefacts. The importance of a sharply marked low limit of frequency range has been shown in Figure 4. In the 5 to 25 cps range, the I-J-K segment may be absent while the pattern becomes identical with that of the total body ballistocardiogram published by Hollis.[27]

In conclusion, the precordial vibrocardiogram in the low frequency range is a subliminal phonocardiogram which presents a definite, constant morphologic pattern, similar to that of tracings of thoracic and body mass acceleration. This similarity leads to the assumption that the tracing represents movements due to cardiovascular forces, probably identical with those registered in ballistocardiography. The experimental results confirm that the precordial vibrocardiogram is less subject to artefacts and errors caused by extracardiovascular factors than the indirect ballistocardiogram.

In spite of a constant time relationship between the waves of the vibrocardiogram and those of other cardiovascular tracings, our understanding of the underlying physiologic factors is not complete. Wiggers refused to admit that changes in intracardiac pressure may be truly reflected in precordial tracings, but the problem deserves further study. The close resemblance between the intra-atrial low frequency phonocardiogram and the intra-atrial pressure tracing on the one hand and the precordial vibrocardiogram on the other (Fig. 6) seems to point out that the latter reveals cardiac pressure phenomena with only minor distortions. The possibility of transmitting changes in intracardiac pressure to the chest wall has also been demonstrated by Schuetz;[10] the precordial phonocardiograms of Schuetz corresponded to his right intra-atrial pressure tracings including the vibrations of the second heart sound. This was explained by him according to the concept that, "the systolic 'jet' affects the whole heart."

Summary and Conclusion

Reproducible, consistent patterns of the precordial "displacement" vibrocardiogram in the 5 to 25 cps frequency range are described. This tracing is identical with that recorded in previous reports with acceleration methods in the same frequency range. It is similar to ballistocardiographic acceleration tracings and to phonocardiograms in the infrasonic band. The method and instrumentation are described. It is concluded that the slow vibrations of the thoracic wall are due to cardiovascular forces, which are closely related to those recorded in acceleration total body ballistocardiograms in the same frequency range.

The time relationship of these vibrations with cardiac events has been studied and the interpretation of the individual oscillations attempted. Simultaneous vibrocardiograms, intrathoracic phonocardiograms, and intracardiac pressure tracings reveal data of interest.

A close relationship was found between changes in intracardiac pressure and precordial vibrations. An understanding of the underlying physiologic basis has been attempted also through the study of respiratory variations of the tracing. A comparative study of different types of microphones and filters underlines the importance of standardized techniques and of employing apparatus with clearly defined physical characteristics.

Acknowledgment

I wish to thank Dr. A. A. Luisada for his criticism and advice.

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Spectral Phonocardiography*

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EARLY IN THE 1940's, Potter and his colleagues at the Bell Telephone Laboratories devised a method for the graphic display of speech sound. They called it "visible speech" and the method was known as sound spectrography. The frequency spectrum of sounds was analyzed and the intensity and temporal characteristics were indicated.

Spectral phonocardiography is the adaptation of Potter's method to the recording and study of cardiovascular sound. In the spectral phonocardiogram (SPCG) time is on the horizontal axis as in most physiologic records. The vertical axis is the frequency scale, not intensity as in the oscillographic phonocardiogram. In the SPCG intensity, amplitude or loudness of the sounds (or rather of the components of the sounds at a given frequency) is indicated by grade of blackness, that is, the intensity of the mark in the recording.

This report is a résumé of five years' experience in the application of this method to the study of clinical and physiologic aspects of cardiovascular sound.[1]

On the technical side, methods for putting the electrocardiogram, pressure pulses, and respirations mark on the record as frequency-modulated signals have been devised. The filter system which provides maximum information has been defined and put in operation. Time-lag and filter distortions have been delineated. Two goals, yet unattained, although significant steps toward them have been taken, are (1) free manipulation of the time scale, specifically "stretching," without undesirable sacrifice of detail in the dimension of frequency; and (2) design of a fool-proof, easily operated and maintained, compact, and economically reasonable unit for general clinical use.

ADVANTAGES OF SPECTRAL PHONOCARDIOGRAPHY

The spectral phonocardiogram appears to have the following advantages over the oscillographic phonocardiogram. The display of the frequency spectrum, its unique feature, is responsible for these three advantages of spectral phonocardiography.

(1) Quality, or timbre, is represented and given physical definition. Musical murmurs are well studied by this method. Differences in quality permit differentiation of pericardial friction murmurs from endocardial murmurs.

(2) Resolution in the dimension of time is improved in the SPCG. Identification of the several components of a complex transient such as the first sound is facilitated. Even if two elements are fused in the oscillogram or even if they cannot be separated at some levels of frequency in the SPCG, they are likely to show separation at certain other levels of frequency.

(3) The wide range of intensity in cardiovascular sound, from the most intense grade 6 murmur to the faintest detectable, is more satisfactorily encompassed in the SPCG. A very loud systolic murmur of aortic stenosis can be accurately displayed in the same record with a very faint diastolic murmur of aortic regurgitation. Spreading out the sounds on the frequency scale has the result that the intensity which must be displayed at a given level of frequency is within the capacity of the method without overloading and distortion.

Potentially, spectral phonocardiography can do all that the ear can do. Indeed, it can probably surpass the ear because (1) it is not wedded to a particular frequency-response curve; (2) it suffers from no "psychoacoustic" impediments, such as fatigue or masking; (3) it provides better resolution in the dimension of time than does the ear; and (4) it produces permanent, quantifiable records.

Three topics will be discussed, partly as illustrations of the use of spectral phonocardiography. I have selected these topics either because of the availability of new observations on

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old phenomena or because the phenomena themselves have been poorly recognized or described hitherto. Obviously no complete coverage of the use of SPCG is provided. The topics are: (1) the functional murmur described by Still; (2) the right-sided Austin Flint murmur; and (3) functional arteriovenous fistula in Laennec's cirrhosis of the liver.

The Still Type of Functional Murmur

In his textbook of pediatrics,[2] George F. Still (1868–1941), the pediatrician of the Great Ormond St. Hospital, London, for whom Still's disease is named, described a variety of musical functional murmurs common in children.

> "And here I should like to draw attention to a particular bruit which has somewhat of a musical character, but is neither of sinister omen nor does it indicate endocarditis of any sort. It is heard usually just below the level of the nipple, and about half-way between the left margin of the sternum and the vertical nipple line...it is systolic, and often so small that only a careful observer would detect it; moreover, it is sometimes very variable in audibility, being scarcely noticeable with some beats and easily heard with others, its characteristic feature is a twanging sound very like that made by twanging a piece of tense string. The bruit is found mostly between the ages of two and six years; as a rule they are brought for some ailment such as a cough, or some indigestion, and the bruit is discovered only in the course of routine examination. . . . It persists sometimes for many months. I have noted it as present in one case for two years. Whatever may be its origin, I think it is clearly functional, that is to say, not due to any organic disease either congenital or acquired. . . ."

All subsequent studies of functional murmurs have indicated that the murmur described by Still constitutes a large proportion of all cases. A murmur answering to Still's description was recorded phonocardiographically in 43.5 per cent of 108 children selected at random by Paulin and Mannheimer.[3] Fogel[4] stated that it is the most frequent variety of functional murmur in children. The category of functional murmur to which the adjective "groaning" was appropriately applied represented 500 of 620 basilar functional systolic murmurs and 120 of 620 apical functional systolic murmurs in the group studied by Lynxwiler and Donahoe.[5] Among 300 children with systolic murmurs judged to be functional, Messeloff[6] found that the murmur was musical in 55 per cent. Stuckey[7] stated that 40 of 145 innocent murmurs were squeaky or musical. The type of murmur under discussion, although possibly not representing a homogeneous group, is certainly frequent in children and is probably the most frequent variety of functional murmur.

Many would disagree with Still's estimate of the site of maximal audibility of the murmur and would place it at the left costal margin in the third and fourth interspaces. The murmur is likely to be more evident when the child is recumbent and after exercise. The murmur is occasionally so loud that ventricular septal defect is suspected.[11]

Oscillographic Phonocardiogram. The characteristics of the Still murmur in the oscillographic phonocardiogram have been described by Harris *et al.*,[8–10] Paulin and Mannheimer,[3] Wells,[11] Dunn,[12,13] and others. These features are (1) a brief gap between the first heart sound and the onset of the murmur; (2) failure of the murmur to extend completely through systole to the second heart sound; and (3) especially and most characteristically, regular, periodic vibrations indicative of musicality.

Spectral Phonocardiogram: All the oscillographic features are well displayed in the spectrogram (Fig. 1). The harmonic pattern clearly demonstrates the musicality of the murmur.[14] A variable amount of noisy element is

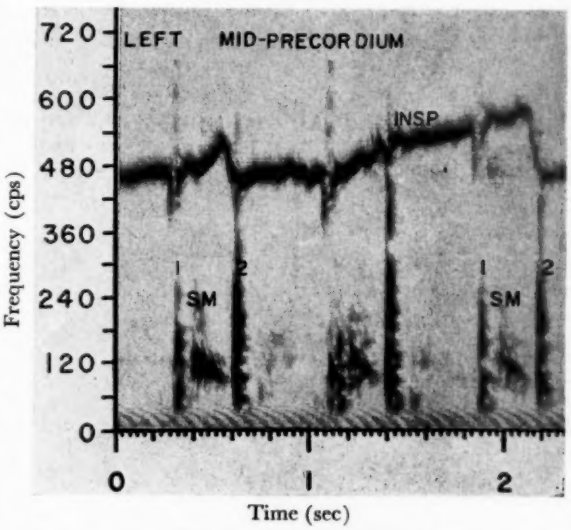

Fig. 1. *The Still murmur.* Left mid-precordium in a 7-year-old boy (J. P., B34751) with apparently normal circulation. Note the brief gap between S_1 and the murmur and between the murmur and S_2. The murmur is somewhat inconstant. Its musicality is represented by the harmonic. Vertical axis represents the frequency scale (cps) and horizontal axis is time (sec).

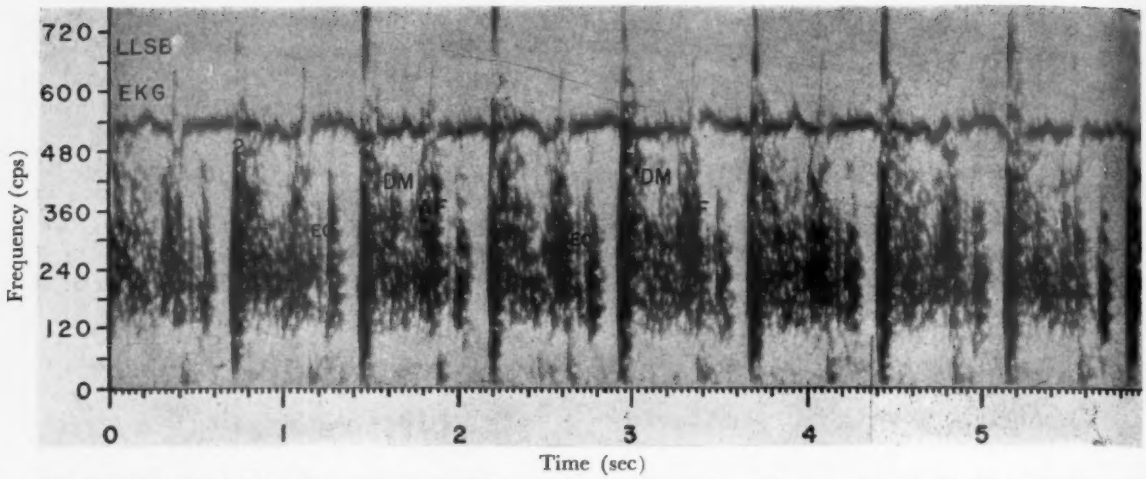

FIG. 2. *Right-sided Austin Flint murmur.* LLSB in inspiratory apnea in a 38-year-old man (V. M., 796346). He had been mildly cyanotic all his life and there was mild clubbing of the fingers. The heart was not enlarged but the pulmonary artery was prominent. The most striking auscultatory finding was a long decrescendo diastolic murmur (DM) beginning with a very loud second heart sound and audible over the entire precordium although loudest at the left sternal border. Increasing with inspiration there was, at LLSB, a loud presystolic crescendo as well (AF). Cardiac catheterization revealed an oxygen step-up at the ventricular level, severe pulmonary hypertension (110/50 mm Hg) and peripheral arterial unsaturation. The shunt, although not measured, was thought to be bidirectional. Mean pulmonary capillary pressure was 4 mm Hg. The patient is thought to fall in the Eisenmenger group. That the pulmonary regurgitation is in part acquired is a possibility. The patient had had at least two episodes of gonococcal urethritis and an episode eight years previously characterized by hemoptysis and joint pains for which antibiotics were given. Bacterial endocarditis on the pulmonary valve may have occurred.

associated with the musicality. Usually only a single harmonic (the fundamental) located between 60 and 120 cps on the frequency scale is demonstrated but one overtone may be shown in the case of louder murmurs.

Mechanism of Production: Experience with the several types of musical murmur suggests that a *sine qua non* for the generation of a murmur which to the ear is musical and in the spectrogram shows harmonic pattern, is the existence of a vibrant member so related to the cardiovascular system, specifically the flowing blood or beating heart, that it is set into vibration in some part of the cardiac cycle. Examples of such vibrant members include the retroverted cusp of syphilitic aortitis, the stenotic valve diaphragm of aortic stenosis, and the wall of critically narrow arteries.

Search for a vibrant member which would account for the Still murmur focuses attention on the cusps of the pulmonary valve and the changes they undergo with trigonoidation.[15] The thin-walled pulmonary artery tends to dilate with ventricular ejection. In extreme instances the pulmonary cusps are pulled taut across a sector of the valve orifice, creating a nearly triangular effective opening. This is what Chisholm[15] referred to as trigonoidation. It is more likely to occur, and is more pronounced with high cardiac output, in a heart which may show some disproportion between the size of the pulmonary valve area and the volume of blood it is required to pass. It is the free margin of the cusps, stretched in the process of trigonoidation, which is suggested as the possible vibrant member in generation of the Still murmur.

The Right-Sided Austin Flint Murmur

The apical murmur described by Austin Flint, which is associated with aortic regurgitation, and is the audible expression of one variety of functional mitral stenosis has not been "explained" to the satisfaction of all. The problem of its genesis need not concern us here. The purpose is to describe the counterpart of the Austin Flint murmur on the right side of the circulation: relative tricuspid stenosis in association with severe pulmonary regurgitation.

Atrial Septal Defect: Relative tricuspid stenosis as evidenced by a rumbling diastolic murmur occurs with conditions of high flow across the tricuspid valve, specifically atrial septal defect and anomalous pulmonary venous return. Dilatation of the right ventricle may collaborate with high flow in the production of this murmur. It usually does not vary in intensity with respiration.

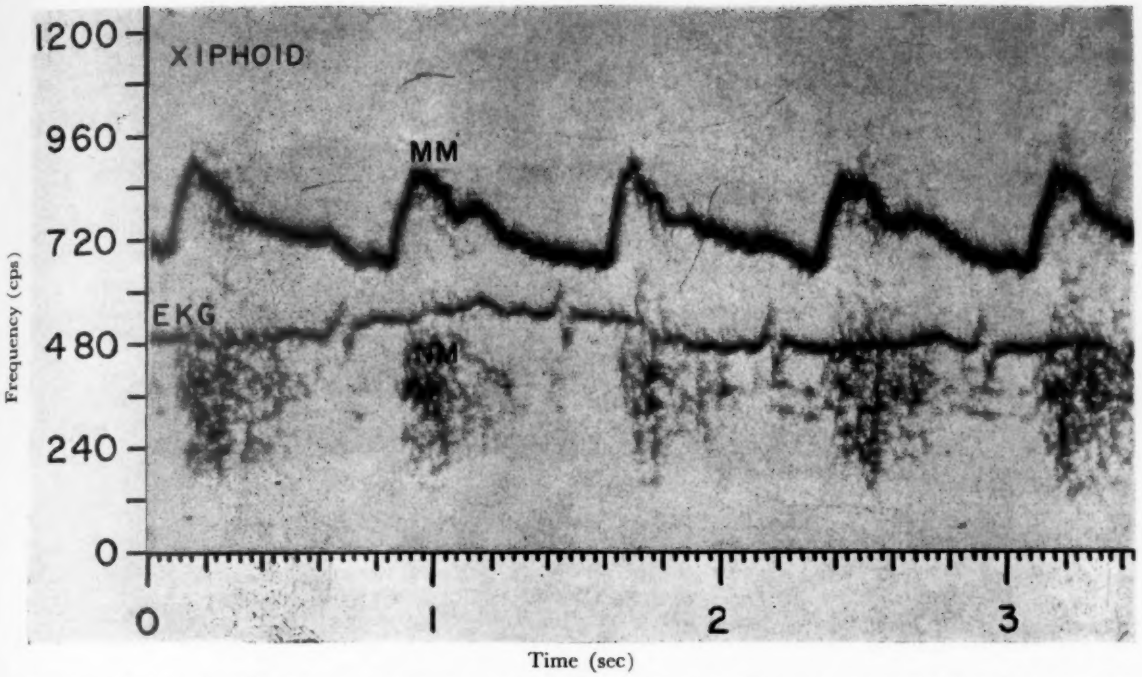

FIG. 3. *Musical Cruveilhier-Baumgarten murmur of arterial type.* In B. S., a 40-year-old man with Laennec's cirrhosis, a dilated, tortuous vessel seemed to perforate to a subcutaneous location in the angle between the xiphoid and the left costal margin. It coursed cephalad along the left sternal border and appeared to perforate into the anterior mediastinum at the level of the fifth intercostal space. The vessel was thin-walled like a vein but had an arterial pulse. Over it was a musical murmur (MM) with the appearance shown here. It was made up of a continuous pure tone (a single harmonic or fundamental) with the shape of an arterial pulse pressure curve. It was located at a higher frequency level than that of most musical arterial murmurs. There was an intermittent noisy murmur (NM) at a lower frequency level. As in other recordings the rise and fall of the EKG indicated inspiration and expiration, respectively. There was no definite change in the murmur with respiration or with the Valsalva maneuver. Localized pressure at either the caudal or the cephalad point of perforation obliterated the murmur. The murmur was audible for a distance of only about two inches from the anomalous vessel. A more conventional venous hum had been present for at least two months before the appearance of this dilated vessel. The arterialized nature of the blood was indicated by an oxygen content of 96 per cent. Increased hepatic arterial blood flow in Laennec's cirrhosis, opening of anatomoses between hepatic arterial radicles and portal vein radicles, and indirectly the development of communications between the hepatic artery and portocaval venous collaterals were suggested. In this case blood was thought to be sluiced fairly directly from a branch of the hepatic artery to a venous collateral draining into the caval system. In essence there was an arteriovenous fistula. It should be stated that this patient had had splenectomy previously. It is my opinion that the anomalous vessel and its murmur were not related to this procedure.

Pulmonary Hypertension: Relative tricuspid stenosis also occurs in cases of severe pulmonary hypertension. Rivero Carvallo *et al.*[16] described a case of this type; the rumbling mid-diastolic and crescendo presystolic murmurs were accentuated during inspiration. MacCallum[17] described a case, from the Johns Hopkins Hospital, of a 29-year-old woman (L. J., autopsy no. 11742) with obliterative lesions of the pulmonary arteries (probably multiple pulmonary emboli) and a mid-diastolic murmur at the apex. In 1935 Wyckoff and Bunim[18] described three such cases and found ten more reported in the literature. One of their patients was a 21-year-old Puerto Rican woman seen in New York with cor pulmonale due to Schistosomiasis mansoni. In this case the diastolic rumble was heard consistently over a period of seven years.

Reubi, Vogt, and Plancherrel[19] described a patient with panarteritis and pulmonary hypertension who had signs of pulmonary regurgitation and right-sided hypertrophy. In addition, there was a diastolic murmur at the apex suggesting mitral stenosis but it was interpreted by the authors as the murmur of relative tricuspid stenosis.

Davis and Andrus[20] reported a case of mediastinal collagenosis in which the pulmonary veins were severely constricted. There was a rumbling apical diastolic murmur probably produced

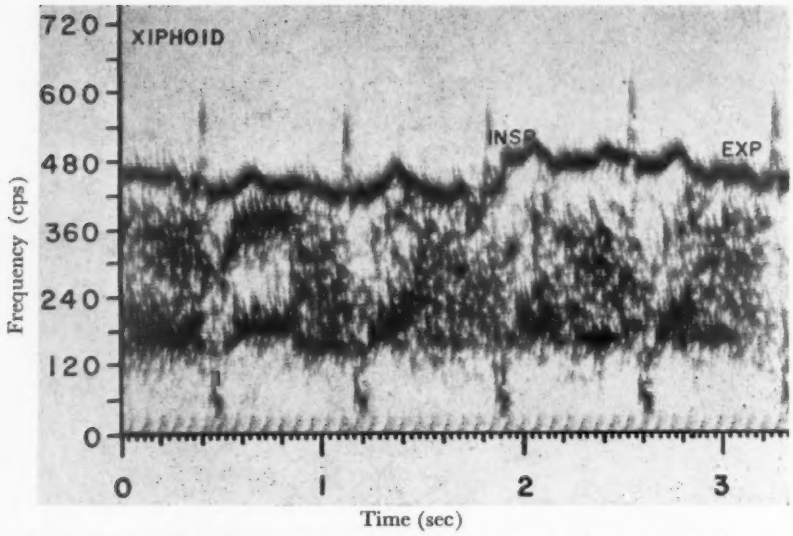

FIG. 4. *Cruveilhier-Baumgarten murmur of more conventional type.* Even here a swelling of the murmur in systole is evident in the harmonic pattern. The patient (V. B., 765876), a 66-year-old woman, had primary biliary cirrhosis. The murmur was well localized to the xiphoid, and often exclusively diastolic, being easily confused with an aortic diastolic murmur. In this recording it is continuous and has a musical quality.

through the mechanism of relative tricuspid stenosis. Brachfeld *et al.*[21] described two patients with primary pulmonary hypertension and a diastolic rumble of relative tricuspid stenosis.

In a recently seen 17-year-old female patient (B. D., 745792, autopsy no. 26655) who had severe primary pulmonary hypertension, a mid-diastolic rumble was heard at the apex. At autopsy there was no septal defect and only extensive pulmonary occlusive arterial disease was present.

Pulmonary Regurgitation: The third type of functional tricuspid stenosis, that associated with pulmonary regurgitation, cannot be separated in most cases from the second type just mentioned since severe pulmonary hypertension is usually present in cases of pulmonary regurgitation. The case presented in Figure 2 is illustrative of the experience with what may appropriately be termed a right-sided Austin Flint murmur. Two other patients with similar findings have come to our attention.

Functional Arteriovenous Fistula in Laennec's Cirrhosis of the Liver

The Cruveilhier-Baumgarten murmur is heard over the venous collaterals, connecting the portal and venous systems, on the abdominal wall of the epigastric area. Laennec's cirrhosis of the liver is the most frequent basis for the portal hypertension responsible for the development of these collaterals. Usually the murmur has characteristics similar to those of other venous hums. We are concerned here with a variety which is continuous, is strikingly musical, and displays in the spectrogram a clear harmonic with the pattern of an arterial pulse pressure curve (Fig. 3). What proportion of all cases of Cruveilhier-Baumgarten murmur the latter type represents is not known; it is not rare, however. The more conventional type is shown in Figure 4.

Mechanism of Production: Experience with the musical uterine souffle, with the partially musical murmur of certain cases of arteriovenous fistula, and with the musical murmur in certain cases of extensive bronchial collaterals with cyanotic congenital heart disease, indicates that when this pattern is observed one can feel confident that there is an unusually large volume of blood being forced through relatively small channels under a head of pressure approaching that in the arterial system and with the phasic changes characteristic of the arterial pressure pulse. The situation is analogous to that in bronchial asthma in which a relatively large volume of a fluid (air) is being forced through relatively small channels and a musical "murmur" is produced. In both situations a "flitter" phenomenon is probably operative. Because

of high axial velocity the walls are pulled in, according to the principle of Bernoulli; opposing forces—the elasticity of the wall and hydrostatic pressure—tend to enlarge the orifice. The net result is a periodic circumferential vibration, or "flitter." The close similarity between the harmonic of the murmur (see Fig. 3) and an arterial pulse pressure curve has its basis in a chain of proportionalities: the frequency at which the generator is driven is proportional to the velocity of flow which finally is proportional to the pressure-head.

Arteriovenous Anastomoses: It seems likely that the "arterial-type" of Cruveilhier-Baumgarten murmur is the functional expression of abnormal communications between the hepatic artery and the portal vein and indirectly the caval system. Herrick,[22] Dock,[23] and others have proposed (1) that there is a relative and probably an absolute increase in hepatic arterial blood flow in Laennec's cirrhosis, (2) that there are abnormally wide arterioportal anastomoses constituting in effect arteriovenous fistulas, and (3) that these contribute to the portal hypertension.

McFadzean and Gray[24] of Hong Kong studied a patient with the arterial type of Cruveilhier-Baumgarten murmur. At laparotomy the murmur could be obliterated by occluding the hepatic artery. At autopsy lipiodol arteriograms demonstrated passage of the contrast medium from the hepatic artery into large branches of the portal vein. Although the authors termed the murmur a "venous hum" in their title, they concluded that it "differed in many respects from that described [by others] and probably had its origin in arteriovenous shunts in the liver."

Summary

The utility and present technical status of spectral phonocardiography are briefly reviewed. By way of partial illustration of the method, the Still type of functional murmur, the right-sided Austin Flint murmur and the musical arterial type of Cruveilhier-Baumgarten murmur are discussed.

It is suggested that the Still murmur is produced through trigonoidation of the pulmonary valve.

It is demonstrated that a presystolic murmur of presumed tricuspid origin can accompany severe pulmonary regurgitation. Other types of "functional," or "relative," tricuspid stenosis are reviewed.

The arterial type of Cruveilhier-Baumgarten murmur is presented as evidence for a functional hepatoportal arteriovenous fistula in some cases of Laennec's cirrhosis, and as direct supporting evidence that abnormally large hepatic-artery-to-portal-vein communications contribute to the portal hypertension of this disorder.

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Frequency Characteristics of Extra Sounds*

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THE FREQUENCY CHARACTERISTICS of cardiac murmurs have been studied with different methods. Cabot and Dodge[1] in 1925, and Williams and Dodge[2] in 1926, were able to differentiate the high and low pitched vibrations of cardiac murmurs by listening through electric filters. Calibrated phonocardiography was introduced by Mannheimer[3] in 1938: tracings were recorded by microphones with a standard type of response for a particular magnitude of vibrations and through filters which excluded certain frequencies. Another method of filtered phono was described by Maass and Weber[4] in 1952. In 1955, Schlitter and Schölmerich[11] described, with the same method, the frequency characteristics of the opening snap of the mitral valve and of the protodiastolic gallop of constrictive pericarditis. In 1956, Luisada and coworkers[5–7] described the method called "selective phonocardiography" which is a modification of the previous ones and permits elimination of some of the sources of error of the other methods. A different device permitted McKusick *et al.*[8] to obtain an accurate measurement of the frequency of any cardiac vibrations (spectral phonocardiography). However, several technical difficulties have limited, so far, the applications of this method.

Few systematic studies have been made in regard to the frequency characteristics of the normal and pathologic heart sounds. We have made a study of the various frequencies of the third and fourth sounds, the opening snap, and the split second sound, both in normal subjects and cardiac patients, by a modification of selective phonocardiography.

MATERIAL AND METHOD

This study was made in 77 subjects. Twenty-five subjects were considered normal; 4 had congestive heart failure; 6 had suffered from myocardial infarction; 16 had rheumatic fever; 13 had rheumatic heart disease; and 13 had various other types of heart diseases. The age of these subjects ranged from 4 to 70 years. A correlation between the frequencies of the heart sounds, the clinical picture, and the age of the patients was attempted. The third sound was studied in 47 cases; the fourth sound, in 23 cases; the opening snap, in 15 cases; and splitting of the second sound, in 10 cases.

Our study was made by using a Sanborn twin-beam connected with a Krohn-Hite band-pass filter according to the scheme of Luisada *et al.*[5] However, instead of setting the high and low pass filters at a distance of an octave or more, both were set at the same figure. This caused an extreme "peaking" of the circuit, so that only a narrow band, extending to less than an octave across the nominal frequency, was obtained. All tracings were recorded with the same degree of amplification, without compensating for the natural decrement of high frequency vibrations.

RESULTS

Third sound: It appeared at a frequency of 20 cps in 45 cases and at 40 cps in only 2 cases. It generally increased in magnitude between 40 and 60 cps, then decreased, but was still recorded up to 60 to 100 cps (Fig. 1). Only in 6 cases was recording possible up to bands from 120 to 200 cps (two of these subjects had heart failure and the others, rheumatic carditis).

Fourth sound: It started at 20 cps in all cases, reached its maximum between 60 and 120 cps and was recorded up to bands from 100 to 200 cps. In 5 cases the final point was found between 100 and 120 cps; in 3 cases, between 140 and 160 cps; and only in 3 cases between 180 and 200 cps.

Opening snap: It appeared at a frequency of 40 cps and reached its largest amplitude in the frequencies of 140 to 160 cps, while in 3 cases,

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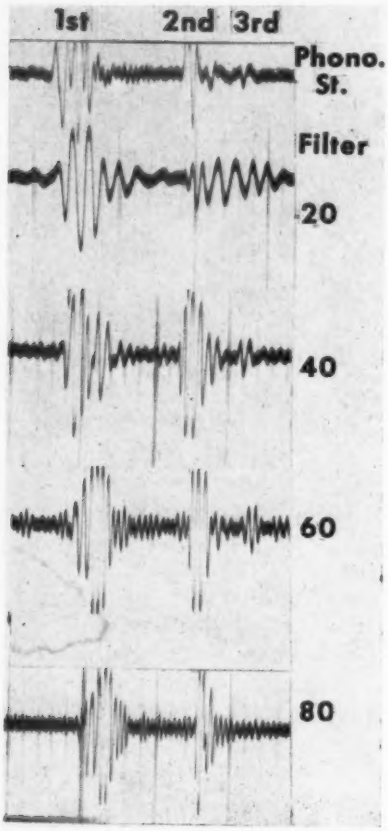

FIG. 1. Normal male subject, aged 13. Upper tracings: Phonocardiogram (stethoscopic) recorded over the midprecordium (fourth left interspace). The subsequent tracings were recorded in the same area at frequencies of 20, 40, 60, and 80 cps. The third sound appeared in the band 20–20 and was still apparent in the band 60–60.

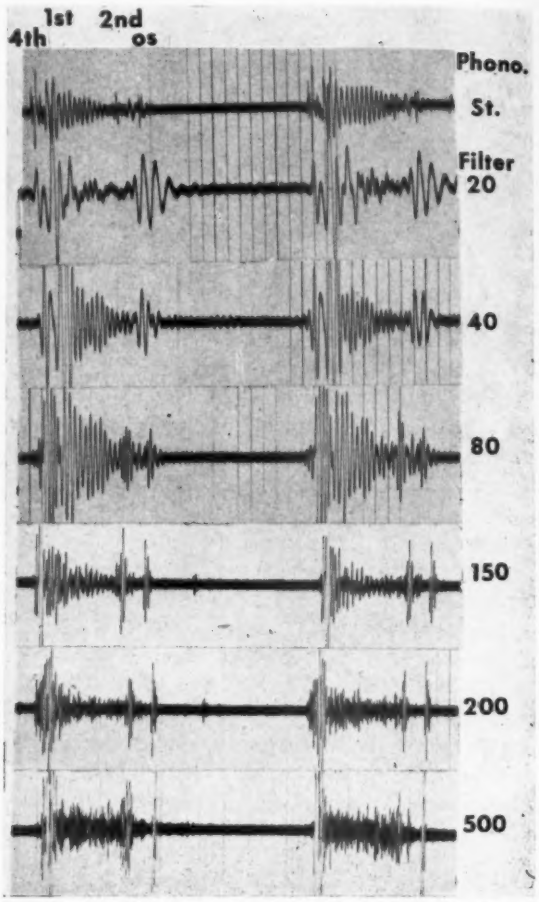

FIG. 2. Male patient, aged 53, with mitral stenosis and insufficiency. Upper tracing: Phonocardiogram (stethoscopic) recorded over the midprecordium (fourth left interspace). The subsequent tracings were recorded over the same area at frequencies of 20, 40, 80, 150, 200, and 500 cps. The opening snap (OS) appeared at low frequencies (40–40) and was still present in the band 500–500. The fourth sound disappeared at the frequency of 80–80.

its end point was found at 140 cps; in the others, it was still recorded in bands respectively from 200 to 500 cps (Fig. 2).

Second Component of the Second Sound: This was recorded in a range extending from 20 to 40 cps as the lower limit and to 300 to 500 cps as the upper limit in all cases (Fig. 3).

Discussion

The third heart sound, a small, dull sound, normally observed in children, has been described in adults, especially in those with conditions causing ventricular strain or overload. Patients with rheumatic and congenital heart disease frequently present this sound. The fourth heart sound is generally recorded in patients with strain of the left ventricle, myocardial damage, or congenital heart diseases with right-to-left shunt.[9–12] The importance of these extra sounds and their prognostic significance is still under discussion.[9]

The presence of an opening snap is considered as being typical of mitral stenosis while changes of pressure, resistances, and flow of the pulmonic system, as well as bundle branch block, may cause splitting of the second sound over the second left interspace.

Differentiation of Extra Sounds by Their Frequency Characteristics: It is important to remember that recognition of a third or fourth sound, evaluation of their clinical significance, and their differentiation from an opening snap or split second sound can be largely based upon an exact graphic determination of their frequency.

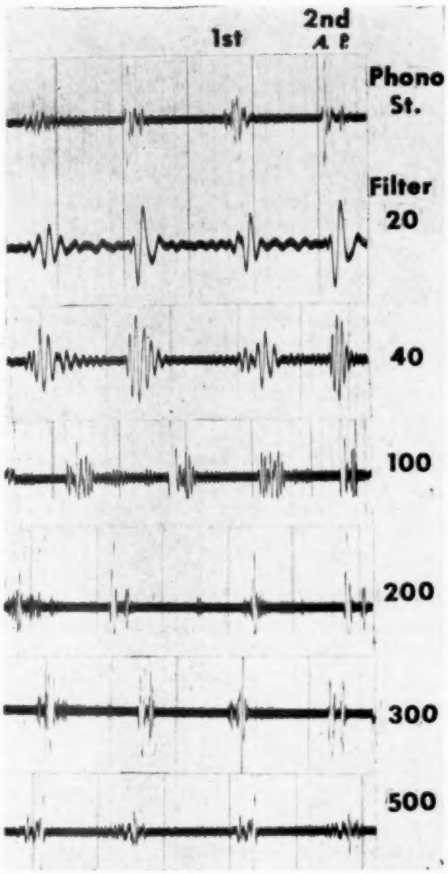

FIG. 3. Normal male subject, aged 10. Upper tracing: Phonocardiogram (stethoscopic) recorded over the pulmonic area (second left interspace). The subsequent tracings were recorded over the same area at frequencies of 20, 40, 100, 200, 300, and 500 cps. The second component of the second sound (P) appeared at the frequency of 40–40 and was still clear at the frequency of 300–300.

Carlgren[13] studied gallops in children with the multiple filter system of Mannheimer and concluded that gallops have a predominant frequency above 100 cps while normal diastolic sounds have a lower frequency. However, Frost[14] was unable to confirm this. Another attempt to measure the frequency of physiologic and pathologic extra sounds was made by Luisada and Roitman[7] in 1949. As their apparatus had a poor response for high frequencies and the differentiation was based on the duration of a sound complex, their attempt was not successful.

Another attempt at differentiating the extra sounds clinically by means of their frequency characteristics was made by Schlitter and Schölmerich[11] in patients with constrictive pericarditis and mitral stenosis by using the method of calibrated phonocardiography described by Maass and Weber.[4] In this method, the filters cut off the frequencies from below upward and recorded the vibrations above 35, 70, 140, or 250 cps. They relied on the natural decrease in amplitude of the cardiac vibrations of higher frequency (so-called "law of the square"; actually, the decrease is more rapid) for giving a natural cut-off from above. The cut-off frequencies were 10 per cent of maximal, or 20 db below the nominal frequencies. This method did not give a sufficient selectivity for the different bands. Moreover, the amplification used (an automatic compensation) was often inadequate. Schlitter and Schölmerich found, with the Maass system, frequencies over 150 to 200 cps for the opening snap of the mitral valve, and lower frequencies (80 to 120 cps) for the third heart sound.

In the series of McKusick *et al.*[8,15] using the spectral phonocardiograph, the gallop sounds were in the frequencies of 80 to 130 cps and the opening snap in the frequencies of 250 to 350 cps. A direct correlation was also observed between the intensity of the sounds and their frequencies: the louder the sounds, the higher the frequencies noted.

In our series of patients, the frequency characteristics of both the third and fourth sounds were similar, in the majority of cases ranging from 20 to 100 cps. In normal subjects, the third and fourth sounds never went higher than 120 cps. In two patients with heart failure and old myocardial infarction and in four patients with rheumatic carditis, the third and fourth sounds also presented higher frequencies (from 140 to 200 cps). However, in the other two patients with congestive heart failure and in the ones with rheumatic disease, no variations from the average range were observed. Therefore, it seems impossible to separate the diastolic sounds of normal subjects from those of abnormal subjects on the basis of maximal frequency. In addition, as the frequencies of the third and fourth sound were in the same range, it was impossible to differentiate them by means of selective phonocardiography.

The opening snap and the components of the split second sound were grossly in the same range of frequencies, even though some higher frequencies were often found for the second sound.

Our data permitted an easy differentiation between an opening snap or a split second sound from a third sound. The former have highest

frequencies which are in much higher range than those of the latter. No particular relation was found between the age of the patients and the frequency of the extra sounds.

Summary and Conclusions

The frequency characteristics of the third and fourth sound, of the opening snap, and of the components of a split second sound were studied in 77 subjects by means of a modification of selective phonocardiography.

No significant and constant differences were found between normal subjects and those with cardiac disease. No relationship was noted with the age of the patients.

It was not possible to differentiate the physiologic third or fourth sound from pathologic triple rhythms by means of sound frequencies.

It was usually possible to differentiate an opening snap or a split second sound from a third or fourth sound by means of the analysis of frequencies.

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Part I of the Symposium on Phonocardiography (Aldo A. Luisada, Guest Editor) appeared in the July issue, Part III will appear in the September issue and Part IV in the October issue.

Clinical Study

Glutamic Oxalacetic Transaminase in Chronic and Acute Peripheral Artery Occlusion

Clinical and Experimental Study*

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GLUTAMIC OXALACETIC transaminase (GO-T) has been found in all tissues and is therefore considered to be an integral component of the cell.[1,2] Following the breakdown of normal cells, it has been demonstrated[3] that 90 to 98 per cent of their GO-T will pass into the blood stream. This observation and the development by LaDue, Wroblewski, and Karmen[4,5] of a comparatively simple spectrophotometric method of determination of SGO-T have resulted in the widespread use of the serum glutamic oxalacetic transaminase test (SGO-T) in the clinical diagnosis of certain types of tissue damage.

SGO-T activity has been studied extensively in a large number of clinical and experimental conditions;[6,8,11–13,16–18] however, there are few observations on SGO-T activity in peripheral arterial disorders. These are for the most part included among large series in which SGO-T levels were obtained in a wide variety of clinical states. Murray *et al.*[7] in their series included two patients with peripheral vascular disease manifesting gangrene of the extremities. Normal SGO-T levels were found in these patients. Among the series of Kattus *et al.*,[14] one patient with thrombosis of the left subclavian artery with normal SGO-T levels (17 to 27 units/ml) is described. In the series of Chinsky *et al.*[6] the only patient described (SGO-T activity of 69 units/ml) manifested gangrene secondary to arteriosclerotic peripheral arterial disease and associated shock-like state. Siekert and Fleischer[10] studied a series of patients with neuromuscular diseases and reported one case of gangrene of the toes in a diabetic patient manifesting an SGO-T activity about two times the normal value. Two patients with bilateral ischemic necrosis of the legs with SGO-T levels of 185 and 50 units/ml, respectively, were recently reported on.[9] Conrad, LaDue and Wroblewski[13,15] state that SGO-T activity is increased in tissue damage due to peripheral arterial disease but they do not discuss specific cases or SGO-T values in these conditions.

The purpose of this study was: (1) to investigate the behavior of SGO-T activity in peripheral arterial disturbances associated with varying degrees of tissue damage, and (2) to evaluate the possible significance of SGO-T levels in certain clinical states (acute myocardial infarction, liver damage, and the like) when associated with peripheral vascular disease.

METHODS OF CLINICAL STUDY

Thirty-five patients with peripheral arterial disease were selected from the Metabolic, Medical and Surgical Departments at the Philadelphia General Hospital. These included 13 males and 22 females ranging in age from 37 to 81 years with an average age of 62 years. These patients were studied clinically to rule out liver disease, acute myocardial infarction and other states which are known to increase SGO-T levels.[2,6,7,10,19] These patients were divided

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TABLE I
SGO-T in Peripheral Vascular Disease

Case No.	Age (yr)	Sex	Diagnosis	SGO-T (unit/ml)
	Group I.		*Patients with Moderate Degree of Peripheral Vascular Disease and No Evidence of Gangrene*	
1	54	M	Thromboangiitis obliterans; cyanosis left toes and foot	17
2	65	M	Arteriosclerosis obliterans; amputation left midthigh; absence of right popliteal and posterior tibial pulse	20
3	62	F	Arteriosclerosis obliterans; intermittent claudication; absence of pulses in right posterior tibial and dorsalis pedis	10
4	63	F	Arteriosclerosis obliterans; intermittent claudication; nocturnal pains in right foot; absence of pulses in right popliteal, posterior tibial, and dorsalis pedis	20
5	81	F	Arteriosclerosis obliterans; intermittent claudication; absence of pulses in popliteal on both sides; thrombosis of bifurcation of aorta (aortogram)	15
6	68	F	Arteriosclerosis obliterans and diabetes; cyanosis of right foot and absence of pulses in dorsalis pedis and posterior tibial	22
7	42	F	Raynaud's syndrome with bilateral cyanosis of first three fingers	25
8	76	M	Arteriosclerosis obliterans; amputation above right knee; nocturnal pain in left leg and absence of pulse in left popliteal	22
9	72	M	Arteriosclerosis obliterans and diabetes; slight cyanosis of toes and absence of pulses in dorsalis pedis on both sides	25
10	59	M	Arteriosclerosis obliterans; intermittent claudication; thrombosis of left popliteal (arteriogram)	20
11	72	F	Arteriosclerosis obliterans and diabetes; cyanosis of left first three fingers; absence of radial pulse	18
12	66	F	Diabetes and arteriosclerosis; cyanosis of right foot and absence of pulses in posterior tibial and dorsalis pedis	25
13	44	F	Raynaud's syndrome; pallor with atrophy of the fingers bilaterally	32
14	67	F	Arteriosclerosis obliterans; no pulses in lower extremities except for right femoral; intermittent claudication	27
15	46	F	Arteriosclerosis obliterans and diabetes; edema and cyanosis of right foot; absence of pedal pulse	18
16	72	F	Arteriosclerosis obliterans; cyanosis and edema of left foot; thrombosis of popliteal artery (arteriogram)	26

into two groups: Group 1, 16 patients with clinical evidence of a moderate degree of peripheral arterial insufficiency without resulting skin lesions or necrosis. The following manifestations of peripheral vascular disease either alone or combined were observed in this group: (1) absence of peripheral pulses, (2) nocturnal and rest pain, (3) intermittent claudication, and (4) alteration in color and a lowered temperature of the skin. Group II, 19 patients with a severe grade of peripheral arterial insufficiency as manifested by gangrene.

In addition to these groups, 10 patients with acute peripheral arterial occlusion were studied. These included nine patients with peripheral emboli and one with acute thrombosis of an arterial graft.

SGO-T was determined on blood samples usually drawn from the antecubital vein. In ten patients of the group with peripheral arterial insufficiency, arteriovenous differences in SGO-T were studied by obtaining simultaneous blood samples from the femoral artery and vein. In three patients who underwent amputation, GO-T concentration was determined (1) from viable muscle taken from the stump and (2) from the perigangrenous tissue of the amputated limb.

SGO-T was determined by the method of Karmen,[5] which in man provided normal values ranging from 8 to 40 units/ml. GO-T activity in the skeletal muscle was determined using the same procedure with homogenated tissue. The samples of muscle taken in the operating room were immediately plunged into ice cold normal saline and homogenated in 0.01 M phosphate buffer solution (pH 7.4) with a Potter-Elvejhem homogenizer suspended in an ice bath.

Results

Peripheral Arterial Insufficiency

GO-T in Serum: SGO-T activity was within normal limits (10 to 32 units/ml, average 21.3 units/ml) in all 16 patients in group I (moderate degree of peripheral arterial insufficiency), as shown in Table I. In 16 of the 19 patients in the second group with evidence of tissue

TABLE I (*Continued*)

Case No.	Age (yr)	Sex	Diagnosis	SOG-T (unit/ml)
		Group II.	*Patients with Severe Degree of Peripheral Vascular Disease and Gangrene*	
17	63	M	Arteriosclerosis obliterans; gangrene of left foot; occlusion of left femoral artery (aortogram)	20
18	52	M	Arteriosclerosis obliterans and diabetes; gangrene of toes and right foot	35
19	60	F	Arteriosclerosis obliterans and diabetes; gangrene of right big toe	22
20	72	F	Arteriosclerosis obliterans; occlusion of posterior tibial on right side and gangrene of foot	15
21	55	M	Arteriosclerosis obliterans and diabetes; gangrene of left foot; occlusion of left popliteal artery and right posterior tibial (aortogram)	20
22	57	F	Arteriosclerosis obliterans; gangrene of 4th and 5th left toes; occlusion of left femoral artery (arteriogram)	20
23	78	F	Arteriosclerosis obliterans; gangrene of right toes	40
24	67	F	Arteriosclerosis obliterans and diabetes; amputation of left first three toes	25
25	51	F	Raynaud's syndrome with gangrene of first three fingers of left hand	30
26	64	F	Arteriosclerosis; gangrene of right foot; thrombotic occlusion of the right femoral artery (arteriogram)	25
27	66	F	Arteriosclerosis obliterans and diabetes; gangrene of first three right toes	25
28	67	F	Arteriosclerosis obliterans; extensive chronic bilateral ulcers	30
29	74	F	Arteriosclerosis obliterans; occlusion of bifurcation of aorta (aortogram); bilateral gangrene of toes	30
30	39	F	Raynaud's syndrome with gangrene of right first three fingers	30
31	71	F	Arteriosclerosis obliterans with gangrene of right foot	20
32	37	M	Thromboangiitis obliterans with gangrene of left big toe; edema and cyanosis of foot	27
33	70	M	Diabetes with generalized arteriosclerosis; bilateral gangrene of feet; occlusion of aortic bifurcation (aortogram)	145
34	81	M	Arteriosclerosis obliterans; gangrene of right foot	110
35	62	F	Diabetes and arteriosclerosis; gangrene of first and second left toes	80

necrosis (Table I), SGO-T was also within normal limits (15 to 40 units/ml; average 25.8 units/ml). It was increased in three other patients in the second group. These subjects presented a degree of tissue damage that was similar to the other 16 members in this group. Other factors were present in these three patients which could explain the rise in SGO-T. One patient, a 70-year-old male with gangrene of both feet, had SGO-T of 145 units/ml and presented a clinical picture of acute myocardial infarction. The other two patients (SGO-T levels of 110 and 85 units/ml, respectively) presented a history of chronic alcoholism, liver enlargement and evidence of serious liver damage which was substantiated by liver function tests.

These findings suggest that the SGO-T activity is not increased in uncomplicated cases of peripheral vascular disease, even in the presence of gangrene of the extent observed in our cases. When a significant increase occurs, it is probably the result of other factors, e.g., damage to the heart or liver.

No arteriovenous differences in SGO-T were observed in the ten cases in which these determinations were made (Table II). There were no clinical cases of acute tissue damage

TABLE II

Simultaneous SGO-T Determinations in Femoral Artery and Vein

Case No.[a]	SGO-T (units/ml)	
	Femoral vein	Femoral artery
17	20	20
18	35	33
19	22	24
20	15	14
21	20	20
24	25	22
25	30	32
26	25	24
27	25	25
29	30	26

[a] The diagnoses of these cases are presented in Table I.

TABLE III

GO-T Determinations in Perigangrenous Muscle Tissue

Case No.	Diagnosis	GO-T per Gram of Wet Muscle		SGO-T (units/cc)
		Muscle of the Stump	Perigangrenous Muscle	
17	Arteriosclerosis obliterans; gangrene of left foot; midthigh amputation	65,900	35,600	20
26	Arteriosclerotic gangrene of right foot; midthigh amputation	64,700	53,500	25
27	Diabetes; arteriosclerosis; gangrene of right big toe; transmetatarsal amputation	67,300	58,400	25

due to peripheral embolism or thrombosis in this series.

GO-T in Skeletal Muscle: GO-T activity in the skeletal muscle surrounding the gangrenous tissue (studied in three patients) appeared significantly decreased in comparison with the viable muscle of the stump, as shown in Table III.

ACUTE PERIPHERAL ARTERIAL OCCLUSION

The site and mechanism of arterial obstruction and the SGO-T levels in these cases 10, 20 and 40 hours after the onset of symptoms are presented in Table IV. Seven of nine patients who had a blood sample taken 20 hours after the acute arterial occlusion showed a significant rise in the SGO-T levels (from 65 to 210 units/cc). These elevations persisted in all five patients who were tested 40 hours after the onset of acute ischemia. The only two patients who did not show any significant rise of SGO-T on the twentieth hour had occlusion of minor arteries in the upper limbs and recovered promptly.

STUDIES IN THE EXPERIMENTAL ANIMAL

SGO-T Following Arterial Ligation in the Hind Limb of the Dog: Dogs weighing between 30 and 40 pounds were anesthetized with Nembutal® (25 mg/kg) and the following experiments were performed:

In the first group of animals (four dogs) arterial occlusion was effected by means of ligation of one

TABLE IV

SGO-T Determinations in 10 Patients with Acute Peripheral Arterial Occlusion

Case No.	Age (yr)	Sex	Diagnosis	SGO-T (units/cc)		
				10 hours	20 hours	40 hours
36	73	F	Midthigh embolus from fibrillating heart; embolectomy 25 hours after onset of pain	70	140	—
37	42	M	Left leg embolus from fibrillating heart in mitral stenosis; embolectomy 35 hours later	—	180	175
38	65	M	Left thigh embolus from atheroma of the abdominal aorta; embolectomy 21 hours later	28	85	—
39	32	F	Right arm embolus from fibrillating heart in mitral stenosis; spontaneous recovery	22	45	45
40	47	M	Acute occlusion of midthigh arterial graft; surgery 20 hours after onset of pain	—	125	—
41	70	M	Embolus in the lower third of the left leg from fibrillating heart; embolectomy attempted 30 hours later	—	65	80 (30 hours)
42	45	M	Embolus in the left arm from fibrillating heart; spontaneous recovery	32	30	75
43	48	F	Right leg embolus from fibrillating heart in mitral insufficiency; embolectomy 15 hours later	45	—	—
44	52	M	Bilateral femoral emboli from fibrillating heart and aortic atheroma; died after attempted embolectomy 24 hours after onset of symptoms	—	210	—
45	66	M	Shower of emboli in both lower limbs; embolectomy performed in the left leg 32 hours after the onset of symptoms	—	85	85 (30 hours)

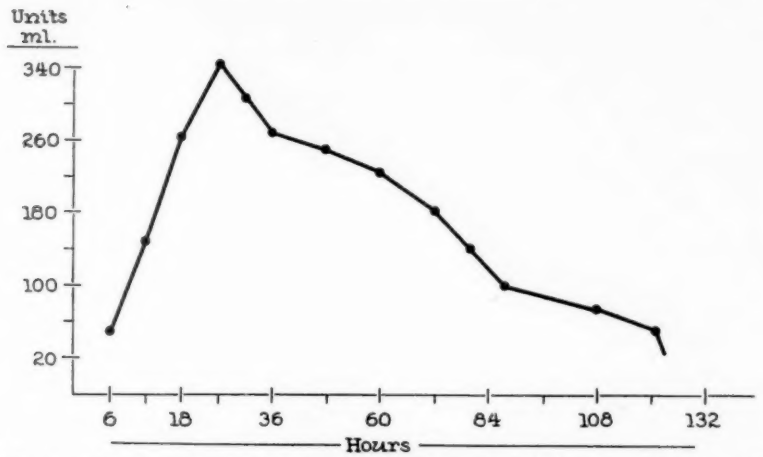

FIG. 1. Effect on the SGO-T levels of embolization of the femoral artery of the dog produced by lycopodium spores. Note that the peak concentration was reached in about 27 hours after embolization (340 units). A significantly high level was maintained for approximately 108 hours.

major artery of the hind limb (femoral, popliteal, anterior, or posterior tibial). On one dog two major arteries were ligated at different times: initially the posterior tibial, and after five days, the femoral artery. In a second group (three dogs) arterial occlusion was carried out by means of embolization with lead balls or lycopodium spores (2 cc of a sterile 10 per cent suspension in normal saline) introduced into the femoral artery immediately below the inguinal ligament. In all dogs, samples of blood were drawn every four hours for the first 24-hour period after occlusion, and then every six to eight hours thereafter, until SGO-T levels returned to control preoperative values.

In the first group of dogs, arteriovenous differences in SGO-T levels were determined across the femoral artery and vein.

RESULTS

In all the animals which had ligation of a main artery of the hind limb, SGO-T levels rose within 10 hours and reached a peak in 48 hours, returning to normal after about 130 hours. An almost identical pattern was observed in the animals which had experimental embolization. SGO-T levels in all cases were roughly correlated to the size of the vessel occluded and the resulting degree of tissue damage. The highest peak (340 units/ml) observed in the animal in which embolization was produced with lycopodium spores was associated with extensive necrosis of the limb (Fig. 1). The SGO-T curve in an animal in which double artery ligation was performed (first the posterior tibial and five days later, the femoral) is shown in Figure 2.

No arteriovenous differences in SGO-T activity in the femoral artery and vein were noted in the animals with acute peripheral artery occlusion.

COMMENTS

The presence of normal SGO-T values in chronic peripheral arterial disease is not surprising since the peripheral circulatory situation is not associated with acute tissue damage and is analogous to that which occurs in chronic coronary artery disease. However, in those patients (group II) with associated gangrene of the extremities it is more difficult to explain the normal SGO-T values. The probable explanation for this may be the fact that in peripheral arterial thrombosis the necrosis develops relatively slowly, resulting in the entrance of GO-T into the blood stream in small quantities and over a long period of time. Under these conditions the SGO-T levels are not significantly affected. Moreover, when a prolonged, gradual ischemia has preceded the necrosis in the extremities, the GO-T content of the tissue decreases (Table II). It should also be emphasized that the gangrene in most of the patients in this series involved the foot (in some instances, bilaterally) in which there is paucity of muscular tissue.

The problem is different in the presence of an acute arterial occlusion in which acute and massive tissue damage results in a sudden release of GO-T into the blood stream and therefore significantly increases SGO-T levels.

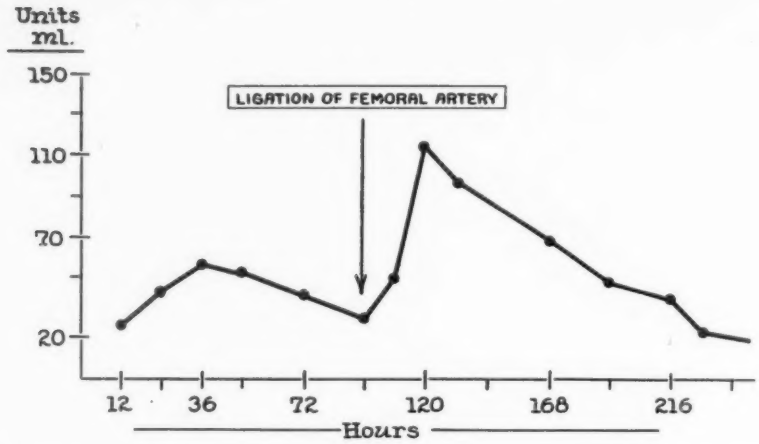

FIG. 2. Effect on SGO-T levels of ligation of the posterior tibial artery in the dog and five days later, the femoral artery. Note the first increase in SGO-T which reached its peak in 36 hours (65 units). The second and higher peak occurred 48 hours after ligation of the femoral artery (115 units). The SGO-T level gradually declined but a significantly high level was observed for approximately six days thereafter.

This situation is analogous to that observed in acute myocardial infarction due to acute coronary artery obstruction.

Acute peripheral arterial occlusion was studied in 10 human subjects, the effects of which are shown in Table IV. Similar studies performed in the dog also yielded results in the expected direction, namely, a significant rise in SGO-T.

It is of interest to explain the absence of significant arteriovenous differences in SGO-T levels as determined on blood taken from the femoral artery and the femoral vein. It was anticipated that the blood drawn from the vein draining the necrotic area would contain a higher concentration of SGO-T. The failure to find a significant arteriovenous difference is similar to the observations in dogs across a systemic artery and the coronary sinus following acute myocardial infarction.[20] This is probably due to the slow rate of entrance of GO-T into the blood stream (in acute myocardial infarction SGO-T requires 18 to 30 hours to reach a peak concentration) and the slow rate of decrease in blood levels after GO-T has entered the circulating blood.

SUMMARY

Serum glutamic oxalacetic transaminase activity was studied in 35 cases of chronic peripheral arterial disease. Group I consisted of 16 patients with a moderate degree of peripheral arterial insufficiency. Group II consisted of 19 patients who had associated gangrene of the lower extremities.

No significant increase in SGO-T level was noted in the first group of patients. A significant increase in the SGO-T level observed in only three subjects in the second group could be explained by the presence of associated acute myocardial infarction (one case) and liver damage (two cases).

Blood samples drawn simultaneously from the femoral artery and the femoral vein showed no significant differences in SGO-T levels.

These findings suggest that in patients with peripheral vascular disease, with or without subacute or chronic gangrene, the occasional appearance of elevated SGO-T levels is probably the result of factors other than the peripheral vascular involvement, usually acute damage to the heart or liver.

Acute occlusion of a major artery in seven of nine patients and in the hind limbs of experimental animals was followed by a significant elevation of SGO-T levels.

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New Methods

Can Routine Electrocardiographic Technique Be Simplified?

Suggestion of a New Method*

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ROUTINE electrocardiographic technique was practiced for over 30 years through the use of the three limb leads suggested by Einthoven. The impossibility to detect electrocardiographic changes taking place in a horizontal plane and along an anteroposterior axis became apparent in the late thirties. Attempts to overcome the inadequacies of the method were made at first by using an anteroposterior lead called lead 4. A new technical era was then started by Wilson and his co-workers with the introduction of the unipolar limb and chest leads. These new leads undoubtedly increased the usefulness of electrocardiography by permitting a point-by-point scanning of the chest surface and identifying the electrical position of the heart better than was possible through the use of the three standard leads.

Following this second stage, it became routine to take 12 electrocardiographic leads, i.e., 3 standard, 3 unipolar limb leads, and 6 unipolar chest leads. To these are often added 1 to 3 right chest leads, the xiphoid lead, 2 posterior leads, and occasionally the esophageal leads. It is apparent that, through the use of so many leads, only an experienced cardiologist is able to obtain accurate information about the various data which are supplied.

Proposals for simplification of the electrocardiographic technique have been numerous.[1,36] It was suggested that the standard leads be abandoned, or that only certain selected chest leads be taken. However, the disadvantages were greater than the advantages and the suggestions were not accepted. If it were possible to obtain from new chest leads all the necessary information regarding the heart, obviously this would permit us to abandon all existing leads with a considerable saving of time and work.

Following the work of many others, we have tried to identify six points on the chest wall which, coupled two by two, would supply three bipolar chest leads, respectively scanning the electrical field of the heart along the three main planes of the body. Following presentation of the method and results, previous work will be discusssed.

METHODS AND MATERIAL

A comparative study of the 12 conventional leads, the vectorcardiographic cubic system of Grishman,[21] the orthogonal system used by Fischmann,[2,3] and our new three chest leads, has been made.

In our system (Fig. 1), six electrodes were placed on the chest in the following way: In the first lead (called X), the negative pole was placed at the crossing of the right parasternal line with a horizontal line passing through the junction of the fifth intercostal space with the sternum; the positive pole was placed on the left over the point 5 of the standard method (corresponding to V_5). In the second lead (called Z), the positive pole was at the same

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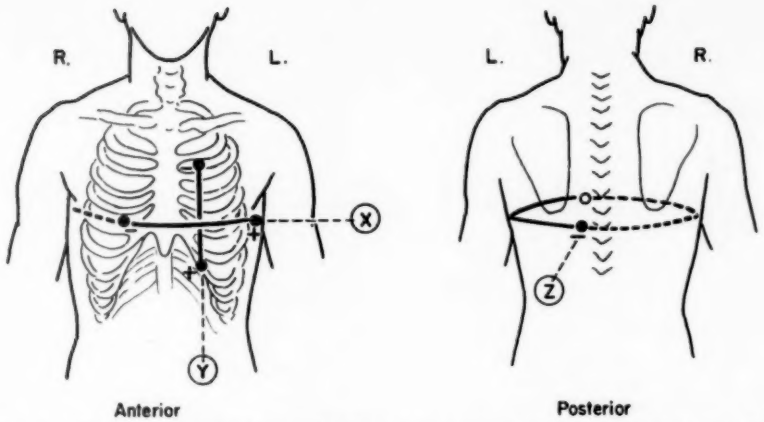

FIG. 1. Placement of the electrodes for the orthogonal bipolar leads X, Y, and Z.

transverse level below point 2 at the left of the sternum while the negative pole was placed posteriorly over point 9 of the standard method. In the third lead (called Y), the positive pole was placed over a point of the left marginal-sternal line at the intersection with a horizontal line passing through the tip of the xiphoid while the negative was placed on the left marginal sternal line, at the second intercostal space.

A system of three orthogonal leads (X or laterolateral; Z or anteroposterior, and Y or inferosuperior) was then formed, each representing a different plane. Their common point of intersection could be considered very near the center of the heart which, as stressed by previous workers[5,6,18,27] should be in a plane passing through the fifth interspace, somewhat to the left of the sternum, and anteriorly in relation to the center of the chest.

The polarity of the leads was such that positive deflections were recorded for cardiac vectors directed anteriorly, to the left, and inferiorly (Fig. 2). For this reason, in normal

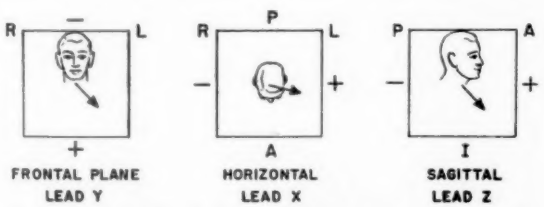

FIG. 2. Polarity of the leads and relationship to the cardiac vector.

cases, positive complexes were recorded in leads X and Y while complexes of an rS type (similar to those obtained in the right precordial leads) were recorded in lead Z. The arrangement of polarity makes it possible to compare lead X with leads 1, aVL, V_5 and V_6; lead Y, with leads 3, aVF, and VE; and lead Z, with leads V_3R, V_1, V_2, and V_3.

The X, Z, Y leads were recorded with a single channel device. The cubic vectorcardiogram according to Grishman was then recorded with a multichannel apparatus* and the loops were photographed on the oscilloscope with a 35 mm camera using Kodak Tri-X films. The orthogonal system of Fischmann was recorded for comparison through use of two channels of the apparatus. All these different tracings were obtained in sequence. A standardization of 1 mV = 1 cm, and paper speeds of 25 and 50 mm/sec, were used.

Twenty-eight normal subjects and 65 patients were studied. Ten of the latter had anterior infarction (2 anteroseptal, 2 anterolateral, and 6 large anterior infarcts); 11 had posterodiaphragmatic or posterior infarction; 1 had anterior and posterior myocardial infarctions; 13 had left ventricular hypertrophy; 12 had right ventricular hypertrophy; 4 had left bundle branch block; 5 had right bundle branch block; and 9 presented evidence of diffuse myocardial ischemia.

RESULTS

NORMAL SUBJECTS

Lead X presented a qR pattern (Figs. 3 and 4). The q was expression of the septal depolarization and corresponded to the initial vector of depolarization that was directed anteriorly and to the right. The R was due to the movement to the left and posteriorly of the ventricular

* Electronics for Medicine.

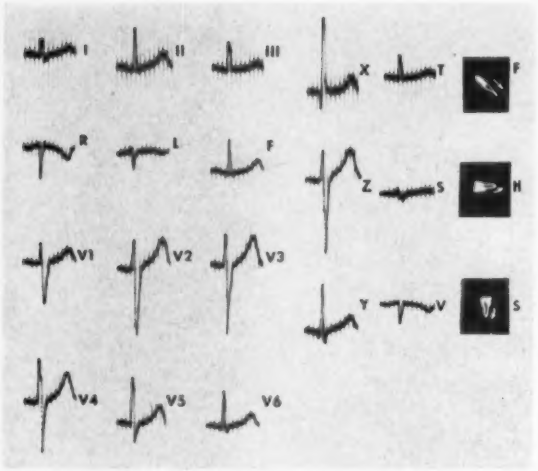

FIG. 3. Normal male, age 21. Normal axis. In this, and in the following illustrations, 1 mV = 1 cm and the film speed is 50 mm per sec. X, Z, and Y are the orthogonal chest leads. T, S, and V are the orthogonal leads of Fischmann and F, H, and S are the cube vectorcardiograms of Grishman. (Film speed 25 mm/sec.)

forces of depolarization and was usually less than 25 mm tall. The relationship between q and R was about 1 : 6 in the majority of cases. The T was positive and had a low voltage in only four cases.

Lead Z had an rS or, in patients with left axis deviation, an RS pattern with positive T. In this lead, r corresponded to the initial vector of depolarization.

Lead Y had normally positive waves, usually

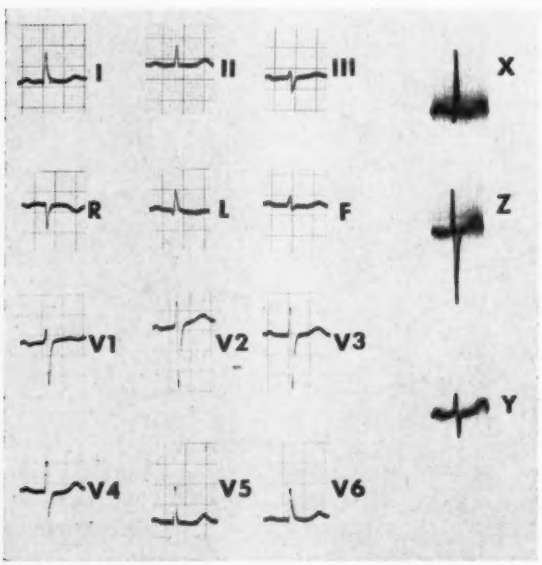

FIG. 4. Normal female, aged 62. Left axis deviation.

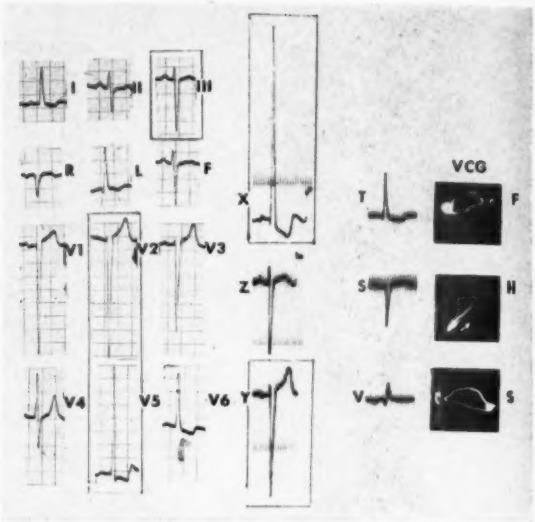

FIG. 5. A 74-year-old male with left ventricular hypertrophy. The patterns in leads X and Y permit recognization of the axis deviation, increased height of R and negative T in X, deep S in Y. These changes, due to the movement of the main electrical forces towards the left and superiorly in left ventricular hypertrophy, are not so evident in leads T and V.

with a large R wave. However, in some cases, the initial and terminal components were recorded as q and s waves of small voltage (less than 2 mm). The T was always positive. In patients with *left axis deviation*, the pattern was

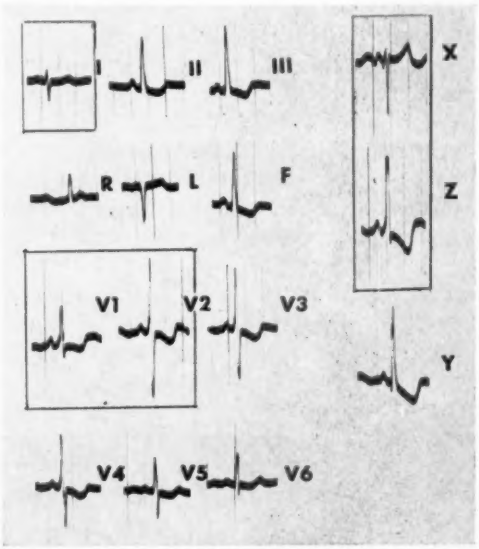

FIG. 6. A 37-year-old female with right ventricular hypertrophy. Leads X and Z show the movement of the main electromotive forces toward the right and anteriorly in right ventricular hypertrophy: deep S in X and tall R in Z.

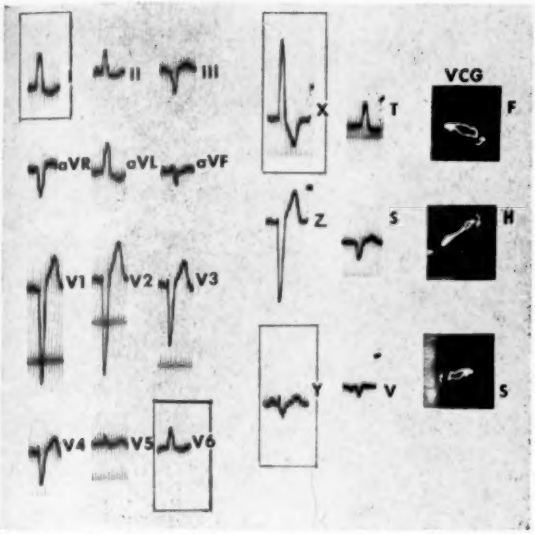

FIG. 7. A 50-year-old female with left bundle branch block. The patterns of peaked X, Z, and Y are significant. Lead X shows a very small q, a tall, notched R, and an inverted T. Lead Z shows a very small q, a tall, notched R, and an inverted T. Lead Z shows a small r followed by a broad S. A broad S is present also in lead Y. In S, there is absence of r and differential diagnosis with an old anterior myocardial infarction would be difficult.

of the RS type because of the horizontal position of the main QRS vector (Fig. 4).

CLINICAL CASES

Left Ventricular Hypertrophy (*Fig. 5*): *Lead X* presented a decrease in the depth of q and an increased voltage of R, which became more than 35 mm. Consequently, a decrease of the q/R ratio was observed. The T was always inverted; the ST segment was depressed. *Lead Z* did not show special changes. *Lead Y* showed an rS pattern because of a posterior and upward orientation of the mean QRS axis. The T wave was in the opposite direction of the QRS complexes. In conclusion, *the most significant changes were observed in lead X* (*latero-lateral lead*).

Right Ventricular Hypertrophy (*Fig. 6*): *Lead X* showed qrs, rS, or QR complexes, due to a shifting of the axis to the right. The T wave was in the opposite direction to the QRS complex. *Lead Z* showed an R or Rs pattern with negative T, due to a forward displacement of the QRS axis. *Lead Y* had patterns similar to the normal or an RS complex with a negative T. *The most significant changes were observed in leads X and Z* (*latero-lateral and antero-posterior leads*).

Left Bundle Branch Block (*Fig. 7*): *Lead X* showed at times a q of smaller voltage than normal followed by a slurred and notched R of normal amplitude; then, a depression of the ST segment followed by a negative T. However, the q disappeared completely in the typical cases. The QRS interval was more than 0.12 sec. In *lead Z*, an rS pattern was observed but r was no higher than 2 mm; S was slurred, and the T was in the opposite direction to the QRS complex. *Lead Y* showed an RS pattern with slurring of S, and T was positive.

Right Bundle Branch Block (*Fig. 8*): *Lead X* showed a qRS pattern; the S wave was slurred and notched; the QRS interval was more than 0.12 sec; the T was positive. In right bundle branch block, the qR pattern could be explained by the orientation of the first and second vector of depolarization, while the terminal vector, oriented anteriorly and to the right, was responsible for the large and notched S. *Lead Z* had an RSR′ complex that resembled the characteristic pattern of right bundle branch block in the right precordial leads. *Lead Y* had positive complexes, similar to the normal but the R wave showed slurring and notching of its contour.

In conclusion, *both in right and left bundle branch block, the most significant leads were X and Z.*

Anterior or Anterolateral Myocardial Infarction (*Figs. 9, 10, and 11*): *Lead X* showed absence of the normal q (or a small and broad q), a normal R wave, and an inversion of T with modifications of the ST segment (in patients with recent infarction). *Lead Z* showed an abnormal complex of the QS type followed by a negative T wave. *Lead Y* had an rS or RS pattern with either diphasic or positive T. The absence of the anterior forces, as a result of the anterior infarction, explains these findings. *The most significant lead was Z* (*anteroposterior*).

Posterodiaphragmatic and Posterior Infarction (*Figs. 12 and 13*): *Lead X* presented a qR pattern similar to that of normal subjects but was followed by an inverted T. *Lead Z* had, in cases of posterodiaphragmatic infarction, patterns similar to the normal; however, in cases of "true posterior infarction," it showed complexes of the RS or Rs type. Lead Y showed, in cases of posterodiaphragmatic infarction a pattern of the QS or Qr type with negative T, similar to the complexes observed in aVF and lead 3. *The most significant lead was, therefore, Y* (*inferosuperior*).

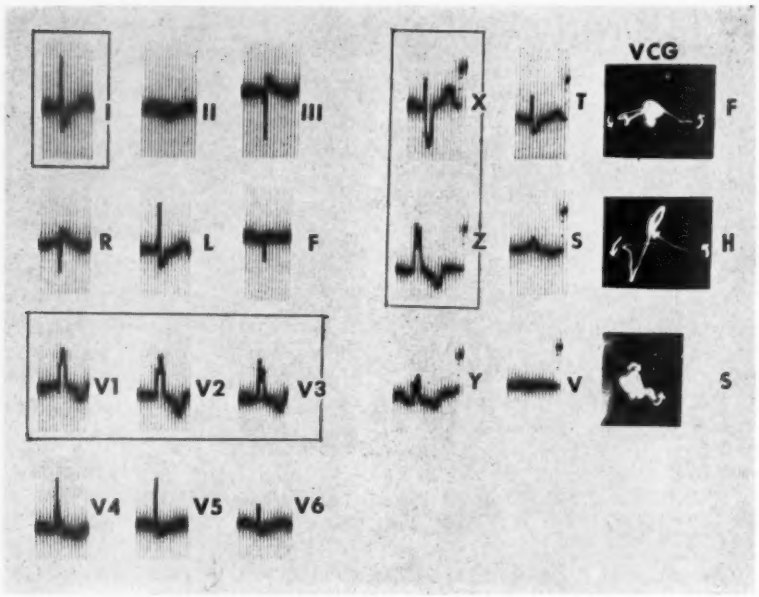

FIG. 8. A 58-year-old female with right bundle branch block. The patterns of leads X and Z are significant. Lead X shows a deep and notched S; lead Z, a wide, slurred R and R′. Similar patterns are observed in T, S, and V.

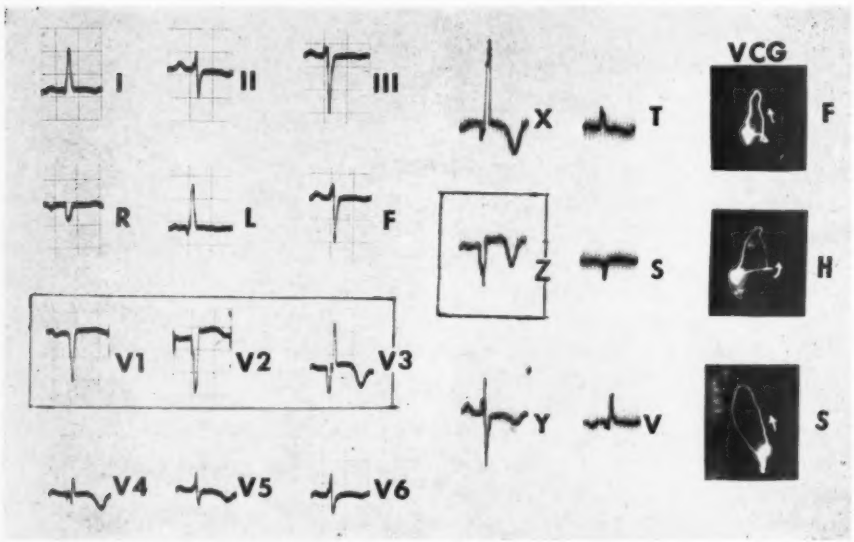

FIG. 9. A 79-year-old male with anteroseptal infarction. Lead X shows a broad and notched q and an inverted T; lead Z shows a QS pattern followed by an inverted T. There is good agreement with the T, S, and V leads.

Myocardial Ischemia (*Fig. 14*): *Lead X* presented a qR pattern similar to the normal, followed by a depressed ST segment and an inverted T. *Lead Z and Y* had patterns similar to the normal, but the T wave was flat or inverted. Changes of the ST segment were also found.

DISCUSSION

Correlations Between the X, Z, Y, Leads and the 12 Standard Leads: In all cases, a good correlation between the 12 standard leads and the three orthogonal leads X, Y, and Z has been observed. In no cases did the 12 leads

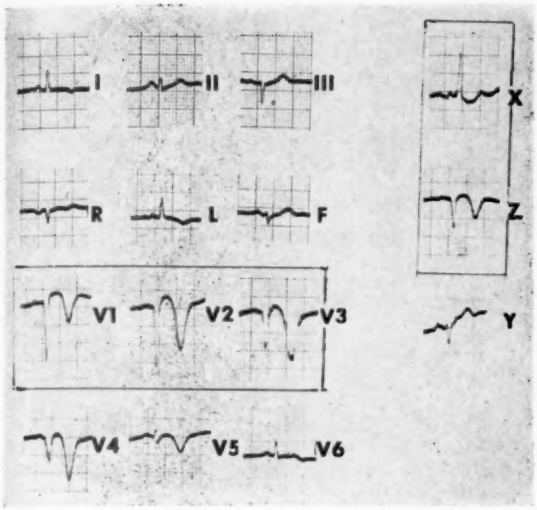

FIG. 10. A 77-year-old male with an old anterior infarction. Lead X shows absence of the normal q and an inverted T. Lead Z shows the typical QS pattern followed by an inverted T.

give more information than that supplied by the new orthogonal leads.

In normal subjects, the X and Z leads, expression of the movement of the cardiac electromotive forces (e.m.f.) over a horizontal plane, are similar in pattern respectively to the left (V_5-V_6) and right precordial leads (V_1-V_3).

In pathologic cases, these two orthogonal leads follow the variations of the e.m.f. in the different conditions: in anterior infarction, the absence of the initial vectorial component explains the negativity of the complex in lead Z and the small size of q in lead X; in right ventricular hypertrophy, the orientation of the QRS axis anteriorly and to the right explains the rS or qrS patterns in X and the prominent R in Z. In left ventricular hypertrophy, the high voltage of R has its explanation in the posterior and leftward orientation of the electrical forces of the heart of such cases. Lead Y, the longitudinal lead, gives the same results as leads 3 and aVF; it was found useful in detecting the inferior infarctions because of the upward orientation of the initial forces in these cases. It was concluded that all the variations of the e.m.f. observed in pathologic conditions may be detected by these three orthogonal leads.

Relationship Between X, Z, Y Leads and the Cubic Vectorcardiographic System of Grishman: The abnormal patterns observed in pathologic cases with Grishman's system had a good correspondence with the orientation of the vectorial loops. The deviations of the initial, medial, and terminal components of the vectorcardiograms corresponded to characteristic variations of the patterns of the X, Z, and Y leads. The latter, however, showed a larger voltage.

Relationship Between X, Z, Y and the Leads Used by Fischmann: The orthogonal system of Fischmann consists of leads recorded according to the cubic system of Grishman with the modifica-

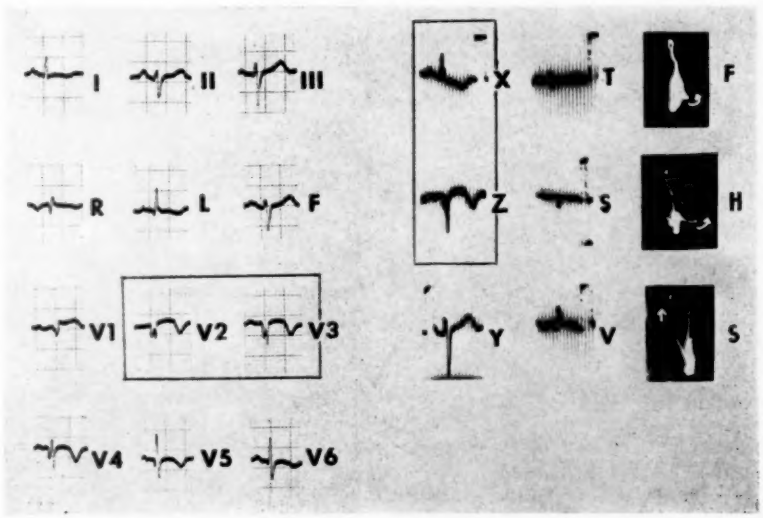

FIG. 11. A 73-year-old male with an old anterolateral infarction. Lead X shows a small q and an inverted T. Lead Z shows the most significant changes: a QS pattern followed by an inverted T. Lead S fails to reveal the anterior infarction and shows an rS pattern.

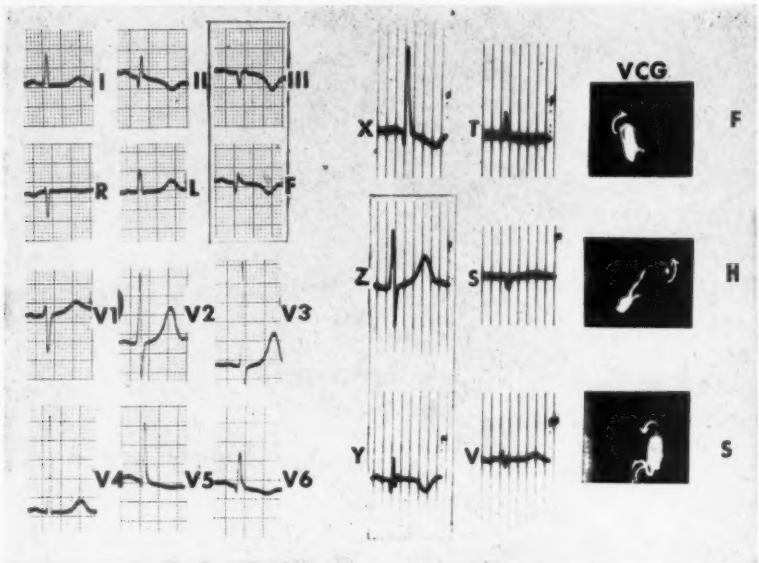

FIG. 12. A 65-year-old male with an old posterodiaphragmatic infarction. Lead Y is the most significant and shows a QRs pattern.

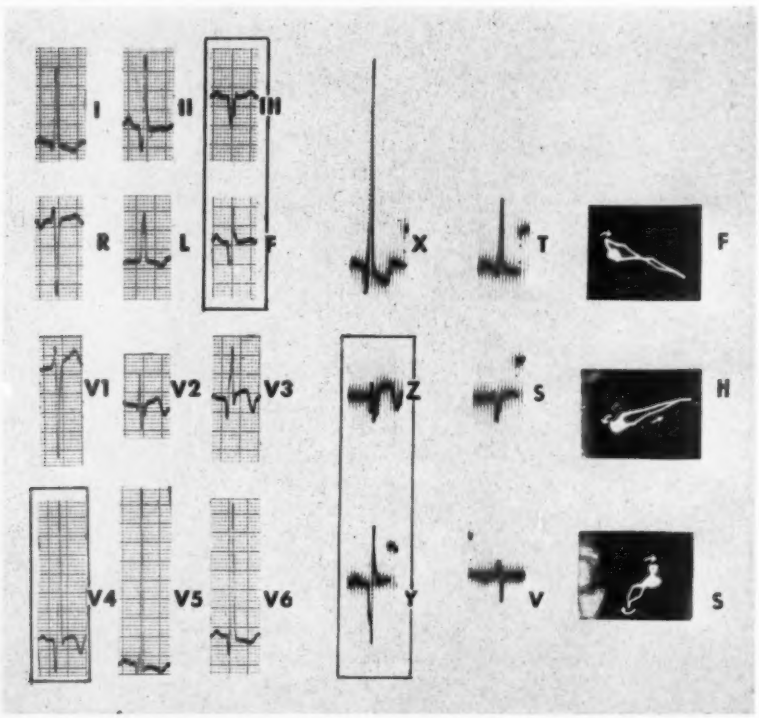

FIG. 13. A 70-year-old male with old posterior and anterior infarctions. Lead X shows a notched and broad q and an inverted T; lead Z shows a QR pattern characteristic of the posterior infarction. Lead S fails to show the Q of an anterior infarction.

tion of Shillingford and Bridgen. According to the latter, a point at the level of the second lumbar vertebra on the right posterior axillary line is chosen as a negative pole for the three leads of the system (T = transverse, V = vertical, S = sagittal).[2,3] This system is useful for vectorcardiographic purposes but, if used as an orthogonal lead system, it reverses the po-

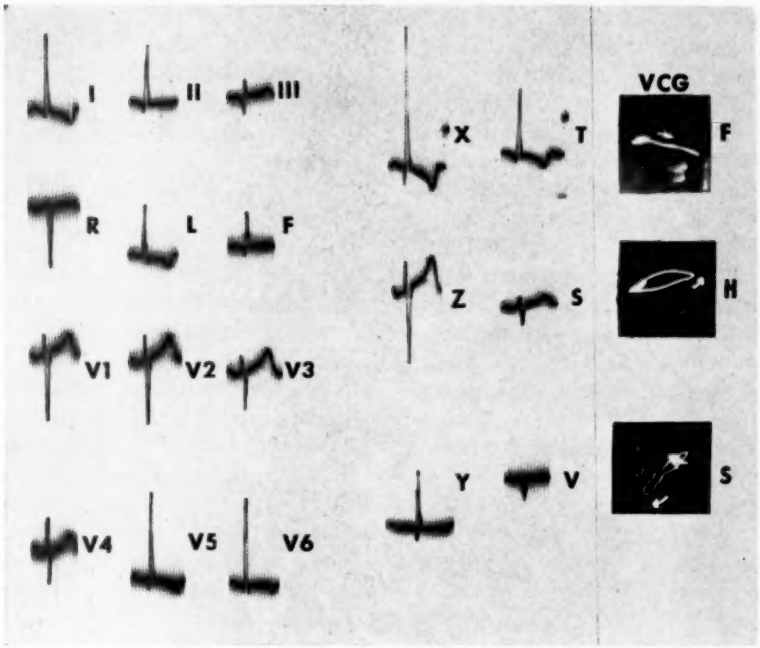

FIG. 14. A 40-year-old female with anterolateral ischemia. Lead X and Y show broad but not inverted T waves and depression of the ST segment

larity of the vertical lead (there is a QS pattern in normal subjects) and may cause difficulty in the identification of posterodiaphragmatic infarctions: positive patterns will be recorded, which have no correlation with the polarity of those found in leads 3 and aVF. This does not occur in our leads, as has been previously shown.

Our vertical lead Y showed a better relationship with the initial and terminal components of the vectorcardiogram than the vertical lead (V) of Fischmann. This can be explained by the different orientation of the effective axis of the two lead systems in relation to the spatial dipole.[4]

The amplification used in our leads was 1 mv = 1 cm. This is less than that required by the cubic orthogonal leads and allows for a better comparison with the 12 standard leads, as well as a better evaluation of the details of the tracings. Moreover, in left ventricular hypertrophy lead X, contrary to the T lead of Fischmann, shows abnormally tall R waves (more than 35 mm high). This significant fact may be explained by admitting, according to the concept of Frank and of Schaffer, that the effective axis of lead L_t (the transverse lead of the tetrahedron system) has a counterclockwise rotation, while the transverse lead of the cubic system is shifted in a clockwise way in its relation to the cardiac dipole. As the electric forces of the heart in left ventricular hypertrophy are oriented toward the left and upward, they will run almost parallel to the transverse lead of the latter and almost perpendicular to the effective axis of the former. Consequently, the tetrahedron system decreases while the cubic system increases the difference between normal e.m.f. and those observed in left ventricular hypertrophy.[4–7]

The pattern observed in left ventricular hypertrophy seems to prove that the position of the axis of lead X is more parallel to the vectorial cardiac forces than that of the transverse lead of the cubic system.

BASIC PRINCIPLES AND CONCEPTS OF ELECTROCARDIOGRAPHY

In the last few years, many publications have attempted to solve some of the points still debated in electrocardiography. The fundamental theories of electrocardiography are based on certain basic principles.

(1) The human body is considered as a volume conductor, having a large size in comparison to the heart, in which the e.m.f. generated by the cardiac muscle is diffused in all directions through a homogenous medium with constant resistances. Experimental work on torso models has stressed the validity of this

concept in order to satisfy at least the necessity of routine electrocardiography.[3,5,8,10]

(2) The electric forces generated by the heart at each instant can be considered like a dipole placed at the center of the conductor. The electrodes of the limb leads have been considered equidistant from the heart and constitute the apices of an equilateral triangle (the Einthoven triangle); Wilson's "central terminal" was built following this assumption.

The exact meaning of the unipolar chest leads also depends upon whether they are expression of the electric activity of the cardiac muscle *below the exploring electrode* or represent the *summation of the electric activity of the entire heart.*

The Dipole Theory: Following experimental studies in dogs, in which the patterns recorded by unipolar leads from the chest wall resembled those directly recorded over the epicardial surface (Wilson *et al.*[11]), the theory that a chest lead reproduced underlying electrical activity was accepted. According to it, projections of the cardiac vectors can be used for the study of the electrical position of the heart and the changes of its electrical axis.[12] Recent work seemed to give support to the first theory.[13] However, experimental work carried out on torso models[5,10] or in human beings with the cancellation method,[14] panoramic vectorcardiography,[15,16] and the axostat,[17] has given support to the second hypothesis, which is based on the principle of the dipole.[18] On this, moreover, are based most vectorcardiographic studies.[19–22] While this theory had already been employed by Einthoven for calculating the electrical axis of the heart on a frontal plane, it was later extended to the horizontal and sagittal planes. In fact, if the e.m.f. of the heart may be represented at each instant by a dipole moving in space, the form and voltage of the currents recorded by any lead (unipolar or bipolar) depend upon the relationship between the axis of a lead and the axis of the dipole in that instant; in other words, from the angle that the lead is forming with the cardiac dipole. If this is true, the precordial leads should be able to reproduce complexes representing the direction and magnitude of the cardiac dipole. According to Duchosal,[19] Frank,[5] and Newman *et al.*,[20] *this concept is valid for any chest lead because the distance of the exploring electrode from the heart is large enough to obtain an application of Einthoven's principle.*

The assumption that the body is a homogeneous volume conductor and that the e.m.f. generated from the heart can be represented by a dipole placed at its center has until now satisfied a qualitative analysis of the cardiac e.m.f. The clinical results have proved reliable, in spite of the manifest imperfections which become more apparent when comparing vectorcardiograms with the 12 electrocardiographic leads.[23]

The heart is not in the center of the chest, but more to the left and anteriorly. Therefore, the dipole will be eccentrically placed and its position varied in different subjects. In addition, the conducting medium is not perfectly homogeneous and the body has an irregular form. These facts are the main cause for the distortion of the "electrical view of the heart" which was found by different authors.[4,10,21,25] Consequently, certain corrections should be applied to the lead axes in order to obtain leads which give an exact quantitative evaluation of the cardiac e.m.f.

Lead Correction Systems: Various attempts have been made to resolve these points. Corrections have been made in the systems of vectorcardiography of Simonson and Schmitt,[26,27] Frank,[28,29] Johnston,[30] Helm[25] and Rijlant.[31] By adding calculated resistances to the leads and using different locations of the electrodes (a variable number, from 7 to 63, was used) on the chest or limbs, these authors tried to obtain the best quantitative evaluation of the cardiac e.m.f.; the leads were corrected according to the shape of the chest, the nonhomogeneous medium, and the eccentricity of the cardiac dipole. These studies are still in a phase of development[23,32–35] and it is not known as yet if they have practical value in pathologic conditions.[35] In addition, they need complex apparatus, a great number of leads, and special care in the placement of the electrodes; all of which may prevent a routine application. On the other hand, authors who believe in the "dipole" theory have tried to evaluate the three orthogonal leads of a vectorcardiographic system. The importance of a comparative study of these leads with the 12 standard leads has been stressed by various authors. A fair correlation was observed with different orthogonal systems in normal subjects.[27,36,44] Even in pathologic conditions, different orthogonal systems have been tried:[37,38,40,41,43] the data observed with the systems of Neheb,[40] Condorelli,[41] Cossio and Bibiloni,[42] and Trethewie,[43] seem to allow a

better identification of myocardial infarction and other pathologic conditions; however, practically, they do not offer significant advantages over the 12 leads of the normal electrocardiogram.[37] More recently, Fischmann,[3] with the use of the three orthogonal leads of Grishman's cubic system,[2] obtained interesting results in the study of patients with left ventricular hypertrophy. He observed a fair correlation between his three leads (V-T-S) and the 12 standard leads and practically obtained with only three leads the same information which was given by standard leads. However, a systematic, comparative study between orthogonal and standard leads in the various positions of the normal heart and in different pathological conditions has not been made as yet.

Advantages of Orthogonal Bipolar Chest Leads: The results presented in this study seem to prove: (1) that the three orthogonal bipolar chest leads suggested and studied by us supply data which are equivalent to those obtained from the standard and unipolar limb leads, and unipolar chest leads; (2) that the patterns revealed by these new leads are significant and permit recognition of the position of the heart and diagnosis of ventricular hypertrophy and myocardial damage; and (3) that these three leads reveal all the various abnormalities of the QRS and T waves caused by ischemia, injury, or necrosis in the various sections of the heart.

The location of the electrodes of these orthogonal leads has been selected empirically following various attempts of placement at the extremes of the major axes which follow the three basic planes of the heart (vertical, horizontal, and anteroposterior).

It is obvious that, if the data which are now supplied by 12 leads can be obtained through the use of only 3 leads, a definite advantage would be derived by saving time and expense. It is possible that these leads may be useful only for a routine electrocardiographic examination of a patient. Even so, their universal application would represent a definite advantage.

Summary

(1) A new system of 3 orthogonal bipolar chest leads has been used in 28 normal subjects and 65 patients.

(2) Details on placement of the electrodes and criteria for analysis of the tracings are presented.

(3) A comparative study between these leads, the 12 electrocardiographic leads, the cubic system of vectorcardiography of Grishman, and the three orthogonal leads of Fischmann has been made.

(4) The three orthogonal leads supply data equivalent to those which can be obtained from the standard and unipolar limb leads, and unipolar chest leads; the patterns revealed by these new leads are significant. They permit recognition of the position of the heart and diagnosis of ventricular hypertrophy, bundle branch block, myocardial damage, and myocardial infarction.

(5) The usefulness of these new leads for routine electrocardiographic examination is discussed.

(6) The findings are discussed according to the dipole theory.

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Experimental Study

Damage to the Brain Following Anoxia in Dogs

Observations Following Asphyxia, Cardiac Arrest and Ventricular Fibrillation*

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A COURSE in cardiac resuscitation was given monthly to surgeons and anesthetists over the past eight years in this laboratory. Observations were made of dogs with the heart in standstill and with the heart in ventricular fibrillation. They were observed for evidence of damage to the brain 24 hours later. These observations are the subject of this report.

EXPERIMENTAL PROCEDURES

Ventricular Fibrillation: Seventy-seven normal dogs were anesthetized with ether. The lungs were inflated by a Rand-Wolfe respirator.[1] The heart was exposed and put into ventricular fibrillation by a small electric current. It was observed for two minutes. The heart was then pumped by hand to circulate oxygenated blood to the brain and myocardium. The heart was readily defibrillated by a shock of 1.5 to 3 amperes. A coordinated beat with adequate blood pressure was restored. The chest was closed and the dogs observed for evidence of damage to the brain 24 hours later.

Cardiac Arrest: Cardiac standstill (asystole) was produced by anoxia in 77 normal dogs. Each dog was anesthetized and the heart exposed as in the preceding experiments. The intratracheal tube was disconnected from the respirator and clamped. The heart was observed until either the aortic pulse disappeared or the heart stopped beating. To correspond with the two-minute period of ineffective circulation in the fibrillation experiments, an additional two minutes elapsed before resuscitation was started. The lungs were well ventilated with oxygen and the heart pumped by hand. A coordinated beat was readily restored and the chest was closed. Observations of damage to the brain were made 24 hours later.

RESULTS

OBSERVATIONS AFTER VENTRICULAR FIBRILLATION

The heart was easily fibrillated by touching it with a small electric current. The movements of the fibrillating heart are coarse at first and then become so fine that they may be scarcely noticeable. Fine fibrillation, however, can be distinguished from complete standstill.

Blood pressure fell to zero within a few seconds because the fibrillating heart does not expel blood.

The myocardium rapidly became cyanosed because the fibrillating muscle fibers rapidly used the available oxygen. The coronary arteries remained pink because the trapped arterial blood did not move out of the arteries in the absence of circulation.

The wink reflex disappeared in one to one and a half minutes and returned several minutes after the heart was pumped.

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The myocardium lost its tone quickly and became flabby. After the heart was pumped for a few moments the myocardium became pink, recovered its tone and the fibrillary movements became coarse. The heart was then easily defibrillated.

After restoration of a coordinated beat an "adrenalin-like" response was frequently seen. This was due to the dogs' own epinephrine released by the stimulus of a stopped circulation.

Cerebral Effects: Observations 24 hours later showed no evidence of cerebral damage in any of these 77 dogs. The motor system was normal. The sensorium appeared normal. The dogs took food and water and responded to attention in a normal manner.

OBSERVATIONS DURING ASPHYXIA AND CARDIAC ARREST

In 58 dogs asphyxiated by deflating the lungs and clamping the intratracheal tube the wink reflex disappeared in an average period of three and a half minutes (range: two to five and a half minutes) and the aortic pulse disappeared in an average period of five and a half minutes (range: two and half to seven and a half minutes). The period of asphyxia was extended two minutes after the aortic pulse disappeared. The average total period of asphyxia was seven and a half minutes (range: four and a half to nine and a half minutes).

The sequence of events after clamping the tube was as follows: The lungs collapsed and became the color of liver. The blood became dark. Respiratory motion became pronounced for a short period. The wink reflex disappeared. The entire animal became deeply cyanosed as did the heart. Cyanosis in the heart was somewhat slower to develop than in the fibrillating heart probably because the heart was using less oxygen, some oxygen was being transported from other parts of the body, and because the heart was able to extract oxygen from markedly reduced blood. The heart rate increased early and then slowed. As it beat more slowly the force of contraction seemed to increase because of diastolic stretching of muscle fibers. The strength of contraction then decreased and the heart dilated. The aortic pulse disappeared and contraction of myocardium was scarcely perceptible. It then stopped beating.

In none of these 77 experiments did the heart go into fibrillation. The electrocardiogram showed nearly normal complexes with each feeble contraction and occasionally remained so after visible pulsation was absent. In the presence of severe anoxia the hearts were in a state of electrical equilibrium. They were stable electrically and remained so until they were pumped by hand two minutes after the aortic pulse disappeared. This manipulation brought in oxygenated blood and created disequilibrium or instability in the cyanosed heart. Then 10, or 17 per cent, fibrillated. Instability was due to differentials of oxygen content.[2] It was a simple matter to defibrillate these hearts.

Cerebral Effects: After recovery from the anesthetic each animal showed evidence of cerebral damage. The motor system was disturbed by varying degrees. Purposeless movements were often seen. Sedative drugs were used. There was mortality of 14, or 24 per cent, within 24 hours from cerebral damage. The manifestations of cerebral damage were more severe in some than in others 24 hours after asphyxia (Table I). No doubt recovery would have occurred in some but the experiment was terminated in 24 hours. This was the first time many of the surgeons taking the course in resuscitation had seen cerebral damage and it was a valuable experience which can be transferred to man.

TABLE I

Cerebral Damage Following Anoxia in 50 Dogs

Duration of anoxia (min)	Damage slight or moderate	Damage severe	
		Survived 24 hours	Death within 24 hours
4.5–4.75	0	0	2
5.0–5.75	2	0	0
6.0–6.75	4	1	1
7.0–7.75	6	10	7
8.0–8.75	5	5	4
9.0–9.50	1	2	0

Discussion

The heart stops beating in ventricular standstill or asystole in the presence of asphyxia. It

does not fibrillate. The electrical condition is that of equilibrium or stability. It is not related to the amount of oxygen available for there was little or none available. The reduction in oxygen supply was equally severe in all parts of the myocardium. A difference in degree of oxygenation in the myocardium creates a state of disequilibrium and this can fibrillate the heart. Differences in oxygenation in the myocardium can create an electrical condition within the heart so that the heart makes its own electricity which can fibrillate it.[3] The end result is the same as touching the heart with an electric wire in which case the source of electricity is outside the heart.

Cerebral damage did not occur when the circulation was stopped for two minutes, as in the dogs with fibrillated hearts. This period of anoxia is safe as far as the brain is concerned if preceded by a period of excellent oxygenation. Cerebral damage did occur when the circulation was absent for two minutes but preceded by progressive anoxia as in the dogs with asphyxiated hearts. The total period of anoxia varied from four and a half to nine and a half minutes and averaged seven and a half minutes. This period of anoxia is not safe as far as the brain is concerned. A similar two-stage form of asphyxia may occur in man in tracheal obstruction or drowning. The effects of a breakdown in ventilation only are about the same as a breakdown in both ventilation and heart beat simultaneously. After tracheal obstruction one might expect oxygen to be picked up by the circulation from other parts of the body and thus slow down the effect on the brain but these experiments did not prove this point. In these experiments cerebral damage occurred in four and a half minutes of asphyxia.

There was no relationship between severity of cerebral damage and the duration of anoxia within the limits of four and a half to nine and a half minutes.

No relationship was demonstrated between the season of the year or the room temperature upon cerebral damage. Williams and Spencer[4] stated that hypothermia applied after restoration of the heart beat reduces cerebral damage. Hypothermia was applied for two or three days and complete recovery occurred in three patients and nearly complete recovery occurred in the fourth.

Summary

(1) Seventy-seven dogs tolerated ventricular fibrillation for two minutes without evidence of cerebral damage.

(2) In 50 dogs with asphyxia for periods of four and a half to nine and a half minutes signs of cerebral damage developed.

(3) Asphyxiation (tracheal obstruction) does not disturb the condition of electrical stability in the heart. When the heart stops beating it stops in asystole. It does not fibrillate. The electrical equilibrium is not influenced by the amount of oxygen received by the heart as long as distribution is uniform.

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Seminar on Ballistocardiography*

Comparison of Ballistocardiographic Systems with Special Reference to the Use of a Jerkmeter†

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In the development of ballistic techniques for the determination of cardiovascular forces, the major problem remains one of evaluating systems and instrumentation. The standardization of techniques and measuring devices must depend on research in the field, and comparisons of methods will be of great importance before widespread clinical utility can be expected.

In the application of high frequency techniques with calibrated instrumentation, there is no doubt that valid data of clinical value can be obtained.[1–3] Systems which measure body motion with the use of a shin-mounted magnet can only sense and record motion accurately at the shin bone. Distortion produced by transmission of cardiovascular force through the body can be considerable. The body produces a partial differentiation of the ballistic signals at frequencies below the natural frequency of the body. In some ways, this could be advantageous to the clinician if the harmonic content were below body resonance. The higher frequency components in the cardiovascular forces become attenuated by the body and both phases and amplitudes are distorted. Since the contribution to diagnosis of frequency components above 10 cps is still unproved, it would be of advantage to use a system which will reproduce these higher frequency components with faithful phase and amplitude relationships. The ultra low frequency bed technique offers this promise above 1 cycle and shou'd merit widespread and meticulous investigation.

Talbot[4] developed a mercury bed which is, for practical purposes, aperiodic, and cardiovascular forces can be recorded with no influence produced by the restoring forces of the body spring. Burger[5] published studies of an ultra-low frequency bed and used some damping to eliminate swing due to respiration. Rappaport[6] has developed an ultra low frequency bed of extremely light weight (5 pounds) which shows promise due to its simplicity and can be used without damping or with a small amount of damping.

Since there is evidence that the ultra low frequency systems can respond to high frequency components (above 5 cycles), the acceleration measurement was differentiated in order to bring out these higher frequency components with greater amplitude. This time derivative of acceleration has been labeled "jerk" to follow conventional engineering terminology.[7]

* This issue contains Part XI of the Seminar on Ballistocardiography (edited by Sidney R. Arbeit, M.D., F.A.C.C.). A schedule of the articles already published may be found on pp. 101–102 of the January, 1959, issue (Vol. III, No. 1). Parts XII and XIII are scheduled as follows: Part XII—Ballistocardiogram in Functional Heart Disease, Nahum J. Winer (September). Part XIII—A Clinician's Approach to the Ballistocardiogram, Edward W. Bixby, Jr., *and* Summary and Conclusions, Sidney R. Arbeit (October).

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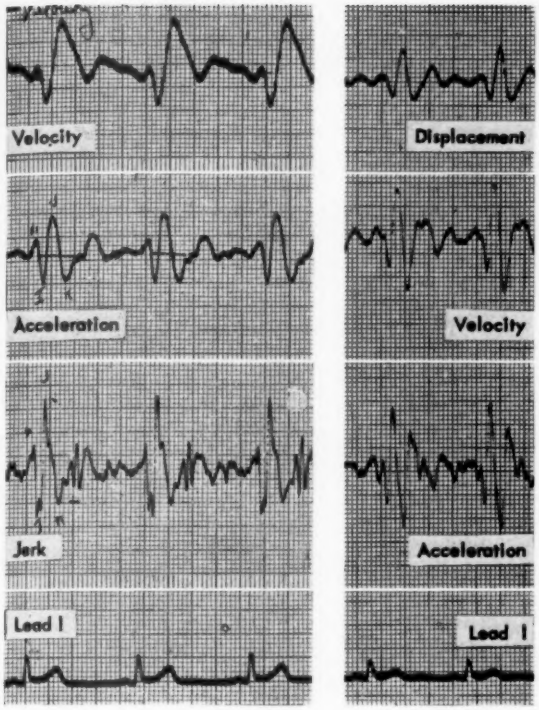

FIG. 1. Case 1. Records of a normal 36-year-old pilot, showing comparison of an ultra low frequency bed ballistocardiogram with the direct body ballistocardiogram.

SYSTEM ANALYSIS AND TECHNIQUE

The ultra low frequency bed recordings used in this study were produced with the use of the Rappaport bed. This bed is constructed of airplane metal and fabric and weighs 5 pounds. The bed is hung by a four-post suspension from a wood framework by means of piano wire 10 feet in length. The natural period of this bed suspension undamped was 0.3 cps. Metal pins with needlepoint sharpness were inserted between one side of the bed and the frame to ensure motion in a head-foot direction with a minimum of lateral motion.

The transducer used in this study was a calibrated bar magnet type.* The magnet was rigidly suspended at the foot of the bed. Displacement, velocity, and acceleration components of bed motion were recorded simultaneously.

Experience in measuring displacement with this system revealed many difficulties due to low frequency resonance phenomena between breathing and natural period of the bed suspension, with consequent marked baseline wandering. This could not be overcome without introducing high damping coefficients with resulting phase distortion. With little damping (0.2 of critical) using cotton between bed and transducer, displacement tracings could be obtained in most cases by suspending respiration.

* Bowen and Company, Bethesda, Maryland.

For these reasons, it is believed that velocity and acceleration recordings would more readily yield reliable clinical data. Also, since this system is capable of responding to higher frequencies, a further signal differentiation would seem to be of value to emphasize higher frequency components. "Jerk" is defined as the time derivative of acceleration. A "jerkmeter" is a transducer which gives an output proportional to jerk.

The jerkmeter used in this study consists of a low frequency piezoelectric accelerometer, the output of which is electrically differentiated. The response is flat ±7 per cent from zero frequency to 18 cps at 21°C. The jerkmeter is calibrated into the recorder so that 1 chart mm of amplitude is equal to 10 mm/sec³. The velocity tracings were calibrated so that 1 chart mm of amplitude is equal to 0.1 mm/sec. The acceleration curves were calibrated so that 1 chart mm of amplitude is equal to 2.0 mm/sec² acceleration.

The jerkmeter has the advantage over most instruments used in ballistocardiography of not requiring a fixed reference point, so that it is inherently isolated from extraneous motions such as building vibrations.

CASE ILLUSTRATIONS

CASE 1. *Normal Individual:* A normal 36-year-old pilot was examined for routine yearly medical certification. History was non-contributory and physical examination showed no evidence of cardiovascular abnormality. Blood pressure was 126/80 mm Hg. The electrocardiogram and double exercise test were normal.

The ultra low frequency bed showed a velocity IJ of 1.8 mm./sec (Fig. 1). The acceleration IJ was 30 mm/sec². The JK jerk (in time related to velocity IJ) was 220 mm/sec³. The direct body ballistocardiogram is illustrated for comparison in Figure 1.

CASE 2. *Coarctation of the Aorta:* This patient was a 23-year-old male weighing 243 lb, with surgically proved coarctation of the aorta. The high frequency (direct body) ballistocardiogram was normal (Fig. 2), but the ultra low frequency system showed a typical coarctation pattern on the displacement and velocity curves (Fig. 3).

Comment: In the analysis of the high frequency ballistocardiogram of the shinmounted type, the variation in displacement amplitudes in normal persons seems to be directly related to the natural frequency of the body. When the natural frequency of the body is high, the ballistocardiogram amplitudes are low, the timing to the H-I-J-K peaks is rapid and the K wave tends to be short. When the natural frequency of the body is low, the ballistocardiogram amplitudes (displacement) are high, the

timing to wave peaks is prolonged and the K wave becomes relatively deeper. These findings are due to the more pronounced effect of resonance in the system plus the fact that body damping is lower in people with low body frequency than in those with high body frequency.

These facts are extremely important when one tries to study coarctation with the direct body methods. The K wave is shortened or absent in coarctation. Since persons with high body frequency tend to show some shortening (Case 2) with low body frequency demonstrated a normal displacement curve on the direct body tracing which obscured the short K wave (see Fig. 2) and a classic coarctation pattern recorded by the ultra low frequency technique (see Fig. 3).

Comparative studies with an ultra low frequency bed show that the coarctation effect is much more pronounced on a displacement curve from this bed than can be found in any other method. It is believed that extremely low frequency systems offer more for quantita-

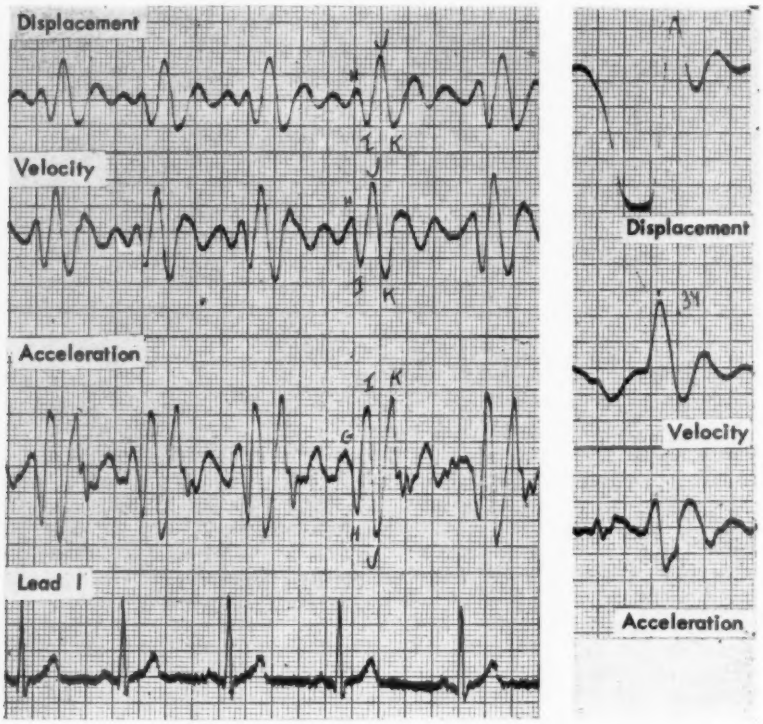

FIG. 2. Case 2. A 23-year-old male with coarctation of the aorta. The high frequency ballistocardiogram is normal, illustrating the effect of low body frequency in obscuring the short K wave of coarctation.

of K waves, false positives could be expected. Since the K wave is deeper in persons with low body frequency, because of the effects of resonance, one would expect obscuration of the coarctation effect in these persons. In 14 cases of coarctation and 2 cases of the Leriche syndrome, shortening of the K wave was significantly greater than that produced by the damping effect of high body frequency. The expected difficulty in differentiating physiologic shortening of the K wave from that in coarctation was, therefore, not great. One patient

tion in coarctation and aortic obstructions than any method used to date. The acceleration curve was proved to be the least valuable for clinical use.

CASE 3. *Aortic Insufficiency:* This was a 41-year-old applicant for pilot certification. There were no symptoms and no past history of rheumatic or scarlet fever. Physical examination revealed a blood pressure of 130/60 mm Hg and a collapsing Corrigan-type pulse. A grade 2 systolic murmur was heard at the apex. A grade 2–3 high-pitched blowing diastolic murmur could be heard at the aortic area and

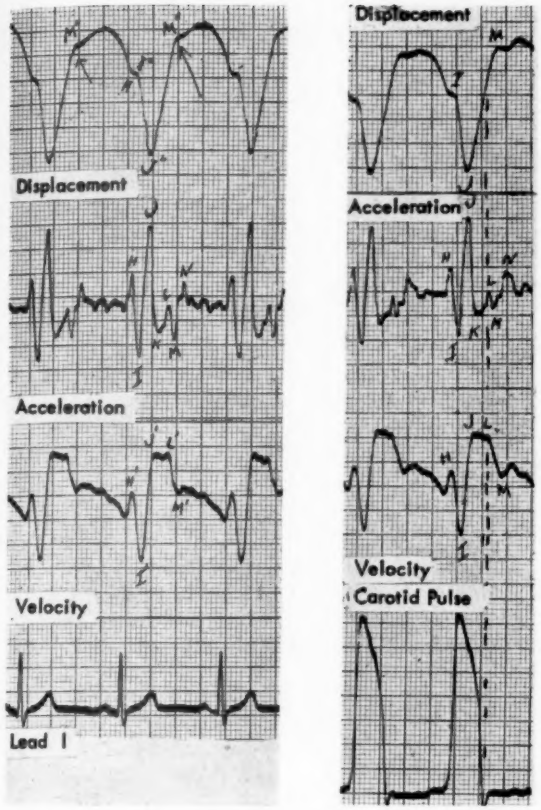

FIG. 3. Case 2. A typical coarctation pattern is recorded with an ultra low frequency system. Note that the displacement curve shows a much larger IJ segment than normal and that the body drifts slowly headward at the M peak instead of returning rapidly to the baseline.

to a lesser degree at the third left interspace. This was recorded phonocardiographically as continuous throughout diastole. Fluoroscopy revealed slight enlargement of the left ventricle with an aortic shadow within normal limits, and normal pulmonary root vascular markings. The electrocardiogram was within normal limits, but R waves in V_4 and V_5 were of high amplitude (28 mm in V_4) with no delay in the intrinsicoid deflection. This suggested some left ventricular hypertrophy. A double two-step exercise test was within normal limits, but the P waves became more prominent, with notching similar to that seen in patients with a P mitrale.

The ultra low frequency bed shows high amplitude velocity curves with IJ of 2.4 mm/sec (Fig. 4). The acceleration curve shows high amplitude on the JK segment with a distinct notch near the baseline. This represents an acceleration of 42 mm/sec^2. A high amplitude notch occurs at the J peak on the jerk tracing. This is seen on the acceleration curve as a barely detectable slope change on the IJ segment. The JK jerk represents a jerk of 370/mm/sec^3 (normal mean = 220 mm/sec^3).

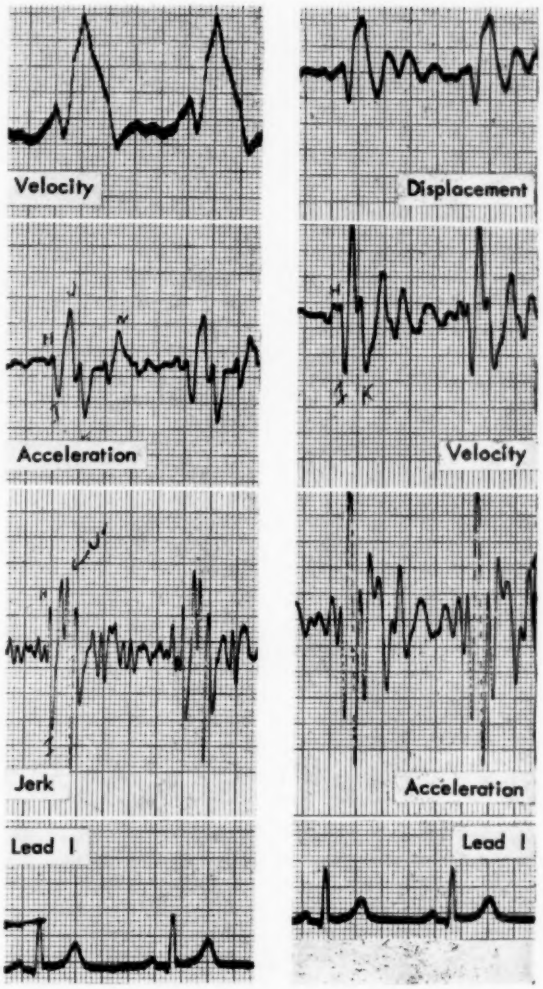

FIG. 4. Case 3. A 42-year-old male with aortic insufficiency and possible mitral valve disease. Note J′ notch on the jerk tracing.

Comment: The J′ notch has been recorded in most cases of aortic insufficiency, using the direct body technique.[8] This case is illustrated because a J′ notch could not be seen in the acceleration traces in either technique. The jerk tracing using an ultra low frequency bed demonstrates this notch at the peak of the J wave. Also, previous harmonic analysis showed that the component was not above 20 cycles. This was true in this case also, since studies with an uncalibrated acceleration transducer flat to above 40 cycles showed no advantage over the illustrated acceleration curve flat to only 18 cycles. In other words, increasing the frequency response of the acceleration measurement to 40 cycles *did not* change the relationship

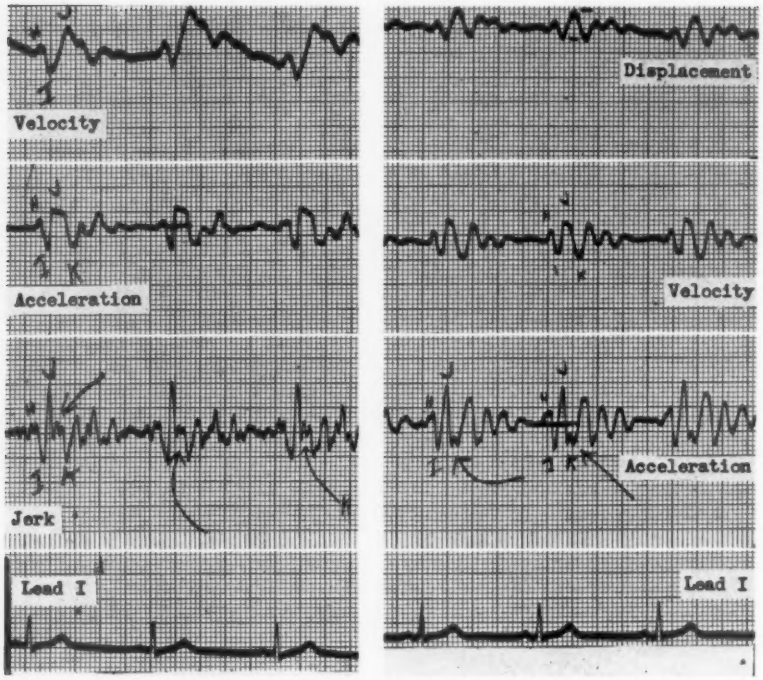

FIG. 5. Case 4. A 52-year-old apparently normal male with ballistocardiographic abnormality only. Discussion in text.

between the acceleration curve and the jerk tracing.

It is of interest in this case that the direct body velocity curve resembles very closely the ultra low frequency acceleration curve. This could mean that the rising response up to 5 cycles due to the natural frequency of the body is producing a signal differentiation before being led into the recording system. This would tend to indicate that harmonics of the ballistocardiogram below 5 cycles is extremely important from the clinical point of view.

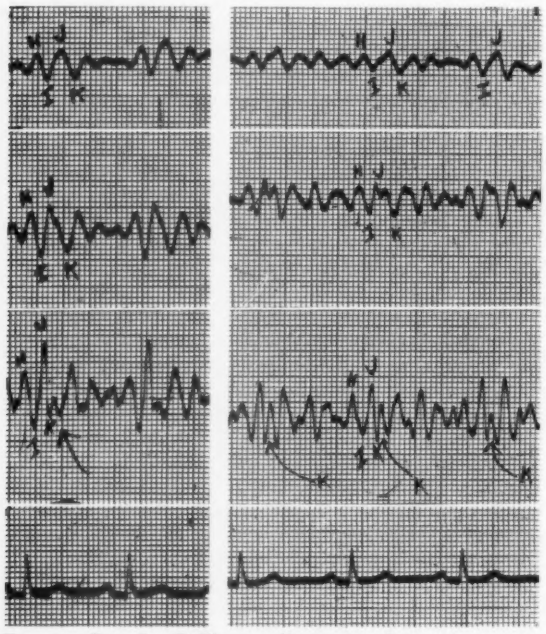

FIG. 6. Case 5. Ballistocardiograms taken prior to the development of myocardial infarction.

CASE 4. *Abnormal Ballistocardiogram Due to Coronary Disease?* This was a 52-year-old applicant for pilot certification. There were no symptoms and physical examination was within normal limits. Blood pressure was 130/80 mm Hg, no cardiac enlargement was detected and the heart sounds were normal. The resting and double two-step exercise electrocardiograms were within normal limits.

The ultra low frequency bed velocity IJ was 1.1 mm/sec (Fig. 5). The acceleration J peak was low and flattened with IJ and JK of 18 mm/sec^2. The acceleration baseline to J peak was 8 mm/sec^2. The jerk recording was of low amplitude with a JK of 160 mm/sec^3 and a double notch at the baseline.

The direct body ballistocardiogram shows low amplitudes of all components, with displacement IJ of 0.0012 inch, and notched acceleration K peak, with a baseline-to-K peak reading of 12 mm/sec^2.

Comment: This case is illustrated to show one of the main problems of clinical ballistocardiography. Clinical experience has in-

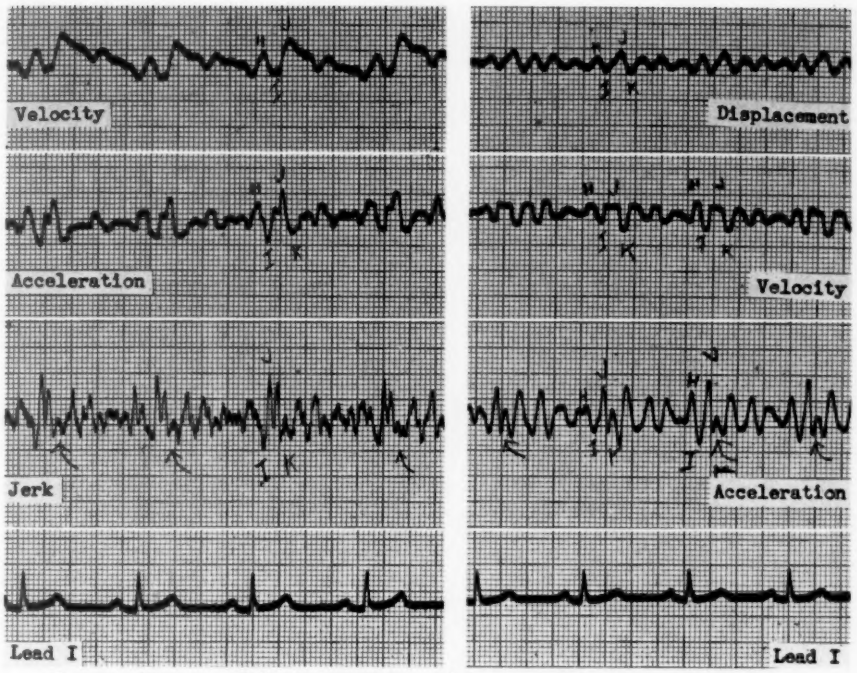

FIG. 7. Case 5. Ballistocardiograms taken six months after myocardial infarction, showing abnormality by both recording systems.

dicated that the direct body ballistocardiographic tracing of the type shown in this case is usually associated with coronary heart disease. The abnormality previously described, notching of acceleration K peak measured directly from the body, seems to be associated with a notched acceleration J peak as measured by the ultra low frequency bed. Low amplitude is found in the acceleration tracing measured by both techniques. These ballistic abnormalities occur, therefore, with both techniques of measurement. From these data, one can conclude that this ballistocardiographic phenomenon is not due to artefact from distortion produced by the body spring system, since the body spring is not acting in the ultra low frequency bed technique of measurement.

Is it possible that these low amplitudes represent changes in cardiac ejection forces and aortic blood flow compatible with the normal aging process? The follow-up statistical data from our laboratory would lead us to believe that these abnormal ballistocardiograms have predictive value. In a study four years ago of 50 normal persons between 40 and 50 years of age, seven, or 14 per cent, showed ballistocardiographic tracings similar to the illustration of the direct body ballistocardiographic tracing in Figure 5. During the past four years clinical evidence of coronary heart disease has developed in four of these seven persons with one death. In none of the 43 in whom the ballistocardiograms were considered normal has clinically evident coronary artery disease developed. With accurate measuring systems and instrumentation, the ballistocardiogram has potential prognostic value. This should rate a high priority for research development.

CASE 5. *Coronary Artery Disease, before and after Myocardial Infarction:* This man was a candidate for medical certification as a pilot. He was examined in July, 1953, for the first time, at the age of 45. He had no symptoms and physical examination was within normal limits. Electrocardiogram and double two-step test were normal. At that time he was included in the group of 50 normal subjects between 40 and 50 years of age in the studies of normal standards. The ballistocardiogram taken at that time was abnormal and was of low amplitude with M patterns in the velocity curve and notched acceleration K peaks (Fig. 6). He was one of the group of seven people found with abnormal ballistic traces mentioned previously.

During the summer of 1954, while on vacation, an episode of epigastric pain was noted, which was followed by thorough gastrointestinal and electrocardiographic studies. It was concluded that some disease of the duodenum existed with a prolapsed mucosa, but the electrocardiogram showed high,

wide P waves in lead 1 and abnormally low T waves in lead 2. On a low calorie diet the symptoms disappeared.

In June 1955 he was again examined for routine pilot certification. At that time he felt well and there were no symptoms. The electrocardiogram and exercise test were normal and no evidence of cardiovascular abnormality could be detected. (The abnormality seen on the electrocardiogram the preceding year was no longer evident.) The ballistocardiogram showed a more pronounced form of the same type of abnormality seen in July 1953 (Fig. 6). The ballistic traces were of lower amplitude, with M patterns in the velocity curve and much more pronounced notching of the acceleration K peaks.

In September 1955, classic symptoms of myocardial infarction developed with electrocardiographic evidence of an acute posterior myocardial infarction for which the patient was hospitalized. Following healing and convalescence, he was studied in February 1956. The electrocardiogram at this time was typical of a healed posterior infarction.

The ultra low frequency bed tracings showed a low amplitude velocity IJ segment of 0.8 mm/sec (Fig. 7). The acceleration tracings were of low amplitude and there was notching of the IJ segment. IJ amplitude was 8 mm or 16 mm/sec^2 and M patterns were present. The jerk IJ was 17 mm or 140 mm/sec^3 with double notching of the JK segment.

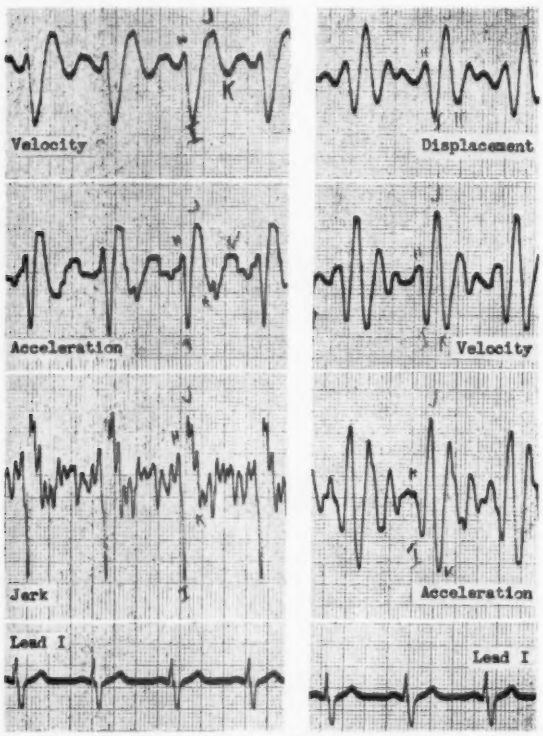

FIG. 8. Case 6. A 22-year-old male with atrial septal defect. Note the high frequency detail that can be seen only in the jerk tracing.

The direct-body ballistocardiogram showed a displacement IJ of 0.0008 inch. The velocity curve was of very low amplitude with flat J peak, and the acceleration curves showed low amplitudes with high amplitude notching of the K peak.

This consistent abnormality could be seen on both techniques of recording (Fig 7).

CASE 6. *Atrial Septal Defect:* This was a 22-year-old applicant for pilot certification, with no complaints but with a history of a heart murmur since birth. There was no functional incapacity or cyanosis. Blood pressure was 115/68 mm Hg. A cardiac murmur was heard best along the left sternal border, of grade 3 intensity and systolic in time with an increased and split P_2. X-ray examination showed increased prominence of the pulmonary vascular markings and of the pulmonary artery segment, with evidence of enlargement of the right ventricle. The electrocardiogram showed right bundle branch block. Cardiac catheterization indicated a left-to-right shunt compatible with a large interatrial septal defect.

The ultra low frequency bed tracing is illustrated in Fig. 8. It shows a deep velocity I wave and a relatively shorter K wave. The mean value for velocity HI segments was 9 mm in our group of normal persons. This compares with 17 mm or 1.7 mm/sec velocity recorded in this subject. The acceleration HI segment shows greater amplitude than is found in normal persons. The jerk HI segment shows extremely high amplitude, with a deep, sharp I wave which can reasonably be attributed to high forces generated in the right ventricle as a result of the left-to-right shunt. The same definition cannot be seen in the leg-mounted ballistocardiogram, which shows only high amplitudes; thus the ultra low frequency bed may yield much more clear-cut data in complex cardiovascular events.

DISCUSSION

The development of ballistocardiographic techniques has been hindered by the complexity of systems and the lack of full appreciation of the technical requirements. The technical studies of Talbot and Harrison[9] have been of considerable assistance, but empiric explorations and clinical comparisons are still vitally necessary for the establishment of clinical utility.

There is little doubt that valuable clinical information can be obtained with the leg-mounted technique. Experience has shown that the ballistocardiographic amplitudes measured from the legs are related to the natural frequency of the body (rigidity of the body spring suspension). Thus, in general, similar internally generated forces will give a lower amplitude for persons with higher natural body frequencies. In other words, the more rigid

the body spring, the lower the ballistocardiograph amplitude, other things being equal, with consequent changes in phase. Any system, such as the pendulous bed, which eliminates the variation produced by the body spring will have advantage since one of the variables (spring distortion) is minimized.

The use of an ultra low frequency bed will pose additional difficult instrumentation problems, since accurate calibration will be absolutely necessary. Instrumentation should respond to much higher frequencies than when used with direct body systems. There is no doubt that the light, pendulous bed can respond adequately to above 30 cycles. At this stage of development it would be desirable to record these components with accurate phase and amplitude, although the clinical significance of responses in these higher frequency ranges has yet to be proved. From the studies to date, with both systems, clinically significant complexes have been found to contain frequency components in the low ranges, mostly below 15 cps. It would seem necessary, in investigating this further, to use a seismic type accelerometer, flat in response to at least 40 cycles, calibrated so that 1 millivolt is equal to 2.0 mm/sec^2 acceleration. This type of accelerometer must be light in weight and not subject to changes in output due to temperature changes or minor adjustments.

If an acceleration curve, flat to 40 cycles, is recorded from a transducer with a ground reference (such as a differentiated curve from a coil and magnet), ambient noise interference such as AC hum and extraneous building vibrations makes evaluation impossible in most environments. At the present time, obtaining accurate calibration has seemed more important than measurement of responses to 40 cycles. Eventually, both calibration and measurement at higher frequencies will be essential in arriving at a more complete understanding of cardiovascular forces.

The use of a seismic jerkmeter may be of assistance in bringing out high frequency components with sufficient amplitudes to be easily studied.

Summary and Conclusions

(1) Clinical experience with an ultra low frequency ballistocardiograph shows that this system is simple to use and capable of responding to high frequencies (above 15 cps).

(2) Recording of the jerk is accomplished with the use of a seismic-type transducer and should be of clinical value.

(3) Comparisons with the direct body ballistocardiograph show that abnormality can usually be demonstrated by both techniques, but the pendulous bed, under certain circumstances, shows components which cannot be demonstrated with the direct body ballistocardiograph.

(4) Illustrative tracings are presented of normal persons, and those with coarctation of the aorta, aortic insufficiency, atrial septal defect, latent coronary artery disease and myocardial infarction.

(5) Further investigation of the clinical significance of components of motion up to 40 cps should be carried out.

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Historical Milestones

Morse K. Taylor on Heart Disease in the Civil War (1867)

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New York, New York

ALL THE READERS of this Journal are aware of the stimulating effect which wars exert upon medicine. This effect is conditioned by the state of medical science at the time of a war and perhaps also by the extent of national involvement. Wars forcibly confront the physician with a large number of patients in a short period of time. This facilitates the recognition and analysis of recurring problems.

The paper which is reprinted herewith is the first of a series of studies on military cardiology that will appear in this Journal. It is planned to present at a later time the famous essay of J. M. Da Costa on the irritable heart of soldiers and the review of neurocirculatory asthenia which was written by Harlow Brooks after World War I.

The text which follows herewith is the work of Morse K. Taylor. It appeared under the title of "Remarks on Heart Diseases as Observed in the Military Service from 1861 to 1865 Inclusive" and was published in the *Transactions of the American Medical Association*, volume 18, pages 137–147, 1867.

Dr. Taylor was born in Watertown, New York in 1823. He served as a cavalry officer during the Mexican War and was discharged in 1848. He then commenced the study of medicine, taking his doctoral degree at the University of Michigan in 1852. Subsequently he held the chair of physiology and hygiene in the medical department of Lind University. He served as army surgeon throughout the Civil War, then entered civilian practice at Keokuk, Iowa. At about this time he was Professor of Physiology and Pathology in the medical department of the University of Iowa. In 1867 he was commissioned Captain and Assistant Surgeon in the United States Army. In 1887, after twenty years of service, chiefly in the West, he was retired from active service because of age. He spent the remaining years of his life at San Antonio, Texas, and died in 1889.

Taylor opens his paper with a statement of the wide differences of opinion which prevailed among Army doctors with regard to the many cases of heart disease that were brought to their notice. His study is an attempt to resolve these difficulties.

* * *

TAYLOR'S "REMARKS ON HEART DISEASES"

It is well known to military surgeons that during the late war a large number of diseases of the heart occurring among the troops were brought to their notice, but in regard to the precise nature of which there was a great diversity of opinion. Some surgeons considered nearly all of them as merely functional in their character, and dependent upon the excessive use of tobacco or coffee; or predicated upon disorders of the digestive system, and particularly of the liver. Others were in doubt as to the nature of these cases; while another class looked upon them as connected with structural changes of the organ itself. The frequency of their occurrence among the patients sent to the General Hospitals at Keokuk, the circumstances attending them, and the contrariety of opinions entertained by medical gentlemen who had treated them, as shown by the diagnosis accompanying their lists, attracted my attention and made it necessary that I should give them my special care.

While making these investigations I observed

some peculiarities, both in regard to symptoms and the relative frequency with which the ventricles were involved, which differed in some respects from the standard works on this class of diseases—the more important one being the structural changes in the right ventricle by dilatation of its walls; conditions denied by many of the surgical staff, and by others scarcely recognized. What I have to present, therefore, will be limited, mainly, to the consideration of these morbid conditions of the right side of the organ. To get at the subject the more readily and clearly, I will present, briefly as I can, a few cases.

Case 1. Joseph Brown, aged 20 years, private in Company "F," 27th Iowa Vol. Infantry, was admitted September 15, 1864, and died November 19th following. When received, he was assigned to the Medical College branch, and placed under the care of Acting Assistant Surgeon G. Sprague. He stated he had been off duty about five months with diarrhoea, but his general appearance did not indicate that he had suffered much from that disease, for he was of full habit, quite vigorous, in good spirits, and able to perform moderate hospital duty.

In about two weeks after his admission he was selected by a medical inspector for field duty, and orders given that he should be sent to his regiment south, with the first detachment. But the post-commander needing some additional men, he was detained and placed on guard-duty for about ten days. Some irregularity of the heart's action had been noticed, but had not received special attention. While on guard-duty he contracted a cold, which presented the ordinary features only of a little bronchial irritation and slight indisposition—not enough to confine him to his bed or prevent his going about. From this he recovered in about three days, and resumed, afterwards, his duties in the ward, which he continued to perform up to within an hour of his death. In the evening of his decease he had been working rather more than usually hard, and, among other things, had drawn a pail of water up to the third story of the building through the "dummie." Some dyspnoea followed, but he ate a light supper at 5 o'clock, as usual, and lay down for the night at 7. An hour after, Dr. Sprague was called hastily to see him, and although attending him in a few minutes, he found him nearly pulseless at the wrist, suffering great distress in the praecordia, breathing laboriously and with pain, and rapidly sinking. In fifteen minutes he was dead.

Post-mortem examination held 40 hours after death. Considerable rigor mortis; body well developed, and moderately full habit; ecchymoses of the face, neck, and pendent portions of the trunk. On opening the thorax an ℥ Jss of bloody serum was found in the pericardium, the coronary vessels very much engorged, and a congested condition of the pericardial membrane existed. The right auricle and ventricle were very much distended, while the left side was nearly empty. On opening the right ventricle, it was found filled with an easily broken down clot, which extended into the auricle and pulmonary arteries. The ventricular wall was scarcely a line in thickness; or about one-sixth as thick as that of the left side. About ℥ xxviij of fluid were taken from the pleural cavities; and quite extensive adhesions existed in the right side, apparently of long standing. Lungs otherwise healthy—very exsanguine. Other organs healthy. Inferior vena cava distended with blood partially fluid.

Case 2. Samuel J. Phillips, aged about 25 years, private in company "C," 1st Wisconsin Heavy Artillery, was admitted August 1, 1864, and died 16th same month. Was assigned to the care of my assistant, Dr. S. Comer, A.A.S., in charge of the Simpson House branch. Soon after his admission, and while examining disabled men for transfer to the *Invalid Corps*, I gave him my special attention. His general appearance was favorable, being in good flesh, and weighing about 160 pounds, full face, and having well-developed extremities. He stated that he had had some months previously a severe attack of measles, that subsequently he coughed, and expectorated very much for some weeks, and that, following this, he began to feel pain in the region of the heart, with at times considerable distress and suffocation on lying down. This increased so that, at the time of admission, on walking fast, or going up a flight of stairs, he suffered much from dyspnoea and palpitation. A physical exploration revealed considerable bronchitis still existing but the pulmonary structures otherwise healthy. The action of the heart, however, was irregular in force and rhythm, oftentimes intermitting; a blowing sound was heard in the right side, with a decided increase in the ordinary cardiac murmurs; the action of the organ was feeble, and the principal impulse was felt at the intercartilaginous spaces, of the sixth and seventh ribs, near the left margin of the sternum, and immediately beneath the free margin of the conjoined cartilages of the lower ribs of the left side, at the junction with the sternum, while the impulse at the apex was very slight. Transverse dullness on percussion was about $4^1/_2$ inches. The lips and extremities were livid, with a decided loss of temperature in the latter, while the face became more or less turgid, according to his position and exercise, the conjunctiva injected, and upon the whole surface of the body, but particularly the anterior portions of the chest, the color was heightened, and the integument pitted on moderate pressure. The jugulars were turgid, but did not pulsate, and the same was true of the veins of the upper part of the chest. A general languor, moderate appetite, tolerably regular

daily evacuations, and high-colored urine were the conditions noted.

On the day of his death, namely, August 16th, he was conversing with his companions in the ward in tolerably good spirits and with manifest improvement from the date of admission, but while sitting on his cot and attempting to rise to go across the room, he staggered, fell, and expired before he could be replaced on his bed.

Post-mortem 36 hours after death. Found the bronchial membranes reddened, with considerable mucus in them, lungs otherwise pale and healthy, but the larger pulmonary vessels moderately gorged with blood. The heart was pale and flabby, the coronary vessels distended, the left ventricle nearly empty, but the right, together with the pulmonary arteries, was filled with a partially organized, easily broken-down clot. The cavity of the right ventricle was fully twice that of the left, and its walls in some places scarcely more than three-fourths of a line in thickness, and presenting the bluish appearance mentioned by some authors upon its pericardial surface. There was considerable vascular engorgement of the liver and kidneys, but the other organs were comparatively healthy.

I could extend these notes to other cases, but as these may be considered as typical of the class I wish to consider, I forbear presenting them and thereby save repetition.

This morbid condition of the heart, however, presents the most interesting features when considered in connection with the antecedent history of the patients. Nine-tenths of them had been preceded by disease of the respiratory apparatus—such as bronchitis following rubeola, pneumonia, or pleurisy, but bronchitis following rubeola taking precedence over the other diseases by nearly double their number jointly. All had been exposed to the severe fatigue and hardships in the Mississippi Valley campaigns of 1862, 1863, and 1864, and had suffered more or less from scorbutus and malarial poisoning. In studying these cases, therefore, I came to the following conclusions as to their origin:—

1st. There had been some impediment to the free circulation of the blood through the lungs at no very remote period.

2d. This impediment arose from very different conditions—one being mechanical obstructions dependent on consolidations of the lung substance, as in pneumonia, or by compression, as in pleuritic effusions; and the other dependent on functional derangements of the respiratory action, as in bronchitis where the introduction of air is more or less interfered with by thickening of the bronchial membranes, or the presence of tenacious mucus; or, on the other hand, from external compression of the thoracic walls by the belts, uniform, and weights borne upon the back and shoulders, thereby preventing the proper aeration of the blood, and with that an arrest of its capillary circulation.

3d. The depressed condition of the muscular structure of the heart as was shown by its feeble action, but further corroborated by the loss of muscular tone throughout the entire system, and which we always find in scorbutic and chronic malarial conditions.

4th. In morbid states of the circulating fluid.

5th. In sudden and undue burdens imposed on the heart during rapid marches or the overwhelming fatigues and excitements of the battle. Many of the patients stated explicitly that the first evidence they had of any ailment in this direction was immediately after such excitements. Indeed, I saw three men sink down, in 1862, out of two companies detailed for some lively skirmishing and quick military movements occupying but an hour or so, from what I apprehended to be this exhaustion of the muscular irritability of this organ, and which was followed by decided functional disturbance for several weeks.

The frequency of the occurrence of these cases may be approximately determined from the following statistics, all of which are obtained from my notes of cases examined by myself, and hence may be considered as reliable as our present means of diagnosis will admit, granting, of course, my possession of sufficient knowledge and skill for their proper application:

The total number discharged from the service on my certificate was	2275
Of these were strictly medical, as distinguished from surgical cases	1366

And of the last number there were 152 diseases of the heart, or 12¼ per cent, classified as follows:

Involving tricuspid valves, with dilatation of right ventricle	24
Involving tricuspid valves, without dilatation being fully determinable	37
Dilatation, without valvular disease	16
Total of right side	77
Involving mitral valves, with dilatation of left ventricle	12
Uncomplicated mitral valvular disease	13
Involving semilunar valves of aorta	7
Hypertrophy of left ventricle, without specification as to valvular disease	26
Total of left side	58

Pericarditis	5
Functional disorders	7
Disease of the heart of doubtful character, and hence not specific as to structures involved, but considered as dependent on muscular debility and anaemia	5
	17
Total discharged for cardiac diseases	152
The total number recommended for transfer to the Veteran Reserve Corps was	735
Of these there were medical, as distinguished from strictly surgical cases	356

And of these latter there were 61 cases of disease of the heart, classified as follows:

Tricuspid valvular disease, with dilatation	8
Tricuspid valvular disease, without dilatation	1
Simple dilatation of right ventricle	13
Total cases of right side	22
Mitral valvular disease, with dilatation	1
Mitral valvular disease, without complication	3
Hypertrophy of left ventricle, without other determinable organic lesions	8
Total of left side	12
Functional disorders of the heart	26
Rheumatic cardialgia	1
Total	27
Making in all	61

Or a fraction over 17 per cent of non-surgical cases thus recommended for transfer.

There were, in addition to the above numbers, about fifty patients suffering more or less from cardiac derangements, that were mustered out of service by reason of the expiration of their terms of enlistment, and of these no particular notes were kept by myself, though one of my assistants, Prof. Magugin, late a member of this Association, kept a record of those in his charge, but which I have not had time to examine fully in detail. About three-fourths of them were of the right side, however, as shown by a casual running over the notes. These were examined by him and myself jointly.

Only a few deaths from this disease occurred under my care. In obstinate cases, the necessities of the service, because of the crowded conditions of the hospitals, together with the desires of the patients, demanded their discharge. Yet I had an opportunity of examining a dozen or more of these fatal cases, and of verifying the diagnosis. Death in nearly all of them was sudden when occurring in the hospital, and under circumstances that indicated an enfeebled and exhausted condition of the organ. In some of them there was a textural change of a fatty character; in all, the heart was very pale and flabby, and showed little or no signs of the rigor mortis.

A large majority of them, however, regained a fair degree of health, and some have become very robust, and retain scarcely any trace of their former trouble. I had some of them belonging to the Veteran Reserve Corps under my supervision for over two years, and hence had a good opportunity to watch their progress; while since the close of the war a few have presented themselves for re-examination, some within the past year. Most of the younger class of patients have gradually improved, but of those past middle life, few have exhibited little, if any, change for the better.

Of the signs and symptoms distinctive of these conditions of the right side of the heart, I can offer but little more than what is presented in standard works on diagnosis. Suffice it to say that the impulse of the organ was feeble; there was irregularity in rhythm, intermitting often, with an occasional *bruit*, and a constant exaggeration of the normal circulatory sounds; a diminished impulse at the apex, but a decided increase at the intercartilaginous spaces of the sixth and seventh ribs, near the left margin of the sternum, and which frequently extended beneath the free margin of the cartilages common to the lower ribs of the left side; increase of the transverse diameter and occasional sharp lancinating pains in the praecordia; a sense of suffocation in lying down, or on exercising; venous pulsations of the external jugulars in about one-half of the cases; engorgements of the liver in about the same ratio. A few had albuminous urine, but in all, this fluid was heavily loaded with the urates, with a specific gravity averaging 1020; lividity of the lips and extremities, flushed face, and in some cases a decided glow over the entire surface of the chest, readily disappearing under moderate pressure, but returning slowly. The integument presented the appearance of passive congestion. In a few cases there was oedema of the inferior extremities, and in others dropsical effusions into the thoracic and abdominal cavities in moderate quantity.

The treatment of these cases was predicated upon the history of the disease and the condition of the patient at the time of admission. One general indication existed in all, namely, to restore the tone of the muscular structures, and

to so regulate the exercise of the patient, as well as his diet, as to secure that end; while employing the ferruginous and bitter tonics, with moderate alcoholic stimulation, immediately after the meals. But nourishing food in moderate quantity, with daily changes in its kind and mode of preparation, light exercise and exposure to the fresh air and sunlight, such as would be secured by placing the subjects on light outdoor duty about the hospitals, was imperative. To allow them to remain idle was quite as objectionable as to require undue labor of them. The employment would keep up their spirits, and prevent their becoming moody and desponding, to which they were particularly prone, and would awaken an interest in surrounding matters.

By this means I am satisfied, that many individuals recovered a good degree of health who, had they been discharged from the service, and been unrestrained in the gratification of their appetites, and with their exercise irregular and unrestricted in amount and kind, and the functions of the heart liable to be overtaxed at any time by their indiscretions, would certainly have died prematurely. I saw a demonstration of this in several patients furloughed, where, after an absence of a month or longer, they returned worse than when they left the hospital.

The alterations effected by this course were such as gradually to diminish the heart's action and increase its force, accompanied by a lessening of its transverse diameter, absence of the intercostal pulsations near the sternum, and venous turgescence of the vessels of the neck and chest, return of the normal impulse at the apex, proper temperature of the extremities, color of the face and trunk, and a gradual diminution of the palpitations and dyspnoea, so that many of them were enabled to pursue their ordinary vocations, with little inconvenience, at the expiration of eighteen months or two years.

The change effected in the heart itself, in these recoveries, I consider due mainly, if not entirely, to a restoration of the muscular structures to a healthful state, thereby reducing its size and cavities by contraction to the normal condition, or very nearly so. In those cases where valvular lesions appeared, especially of the right side, the changes effected were of a twofold character, e.g., those affecting the blood, and those connected with the diminished size of the ventricle whereby the tricuspid valves could more perfectly close the auriculo-ventricular orifice. It was evident, in the second case cited, that the *bruit* in the right side was dependent on imperfect closure of this opening, for the valves were normal in structure, and the post-mortem made it very apparent that when the ventricle was distended they were incompetent to prevent the regurgitation of the blood.

So far as the blood was concerned, some interesting features were observed in the post-mortem examinations of these and other cases. Many of them were associated with chronic diarrhoea, sometimes in its severer, but more generally in its milder forms. In nearly every case where an examination of the heart was made in death from chronic diarrhoea, fibrinous concretions, more or less organized, were found attached to the *chordae tendineae* and auriculo-ventricular valves. The most firmly organized were, in a majority of cases, observed in the left ventricle. Still, those on the right side were sufficiently organized, and so firmly attached in many instances as to require some considerable force to remove them. I apprehend, therefore, that some of the causes which I have classified as "disease of the tricuspid valves, without determinable dilatation," were due to such causes. Recoveries in these cases, then, involved either the forcible detachment of these connections as embodied, and their transmission to the pulmonary circulation, or of their gradual solution "*in situ*." I incline to the belief that the latter mode of removal was not uncommon. The generally improved condition of the blood, and the mechanical action of that fluid on these substances, in accordance with the laws of attrition, give support to the proposition.

* * *

Comments

It must be confessed that Taylor's paper makes an unsatisfying impression despite the fact that the individual cases seem to have been studied carefully and described clearly. In fatal cases autopsy was performed, although without microscopic examination. In a few patients the author was able to make follow-up observations. In the tradition of the French school, which was influential in nineteenth-century America, Taylor also subjected his entire case material to statistical analysis. Yet despite Taylor's obvious diligence, which is demonstrated by his collection and analysis

of first-hand data, the reader is conscious of disappointment. Probably this is due to the author's failure to define his problem with sufficient precision and his failure to reach clear-cut conclusions.

Taylor was interested especially in cases of heart disease in which no definite structural flaw could be detected or no distinct cause could be found. It seems probable that some of his patients had genuine myocarditis. Others undoubtedly had rheumatic endocarditis, as is clear from the final paragraph of his article. Others probably had arrhythmias which nowadays can be described with precision but which even now cannot always be explained. Most interesting is the complete absence of any mention of coronary disease and congenital heart disease.

Two cases are reported in detail. The first is that of a twenty-year-old soldier who had had a variety of infections, including five months' of diarrhea and a brief respiratory ailment. He also had had some type of arrhythmia. On the last day of his life he was on duty, worked hard, and had dyspnea. In the evening he suffered severe precordial distress, was found to be nearly pulseless, and soon died. Autopsy revealed the presence of pleural effusion, dilatation of the right side of the heart, and "a congested condition of the pericardial membrane." The cavities of the right side of the heart and the lumens of the pulmonary arteries contained material which was probably postmortem clot. The reader may exercise himself by trying to decide whether the patient had had rheumatic fever or viral pericarditis or even pulmonary embolism, and these do not exhaust the list of possibilities. Similar obscurity surrounds the second detailed case report.

In surveying his entire series Taylor observed that nine-tenths of the patients had had respiratory infections, especially bronchitis following rubeola. All had undergone severe hardships during the military campaigns of 1862–1864, and many had had scurvy and malaria. He concluded that these patients had in common an impediment in the pulmonary circulation; he lists a large number of conditions which might cause this. He was impressed especially by evidence of dilatation of the right side of the heart. He considered this condition to have occurred much more frequently than was generally recognized, yet he is frank to state that some of his colleagues disagreed with this opinion. He also found evidence of a depressed condition of the myocardium.

Taylor's work was an effort to analyze large groups of miscellaneous cases, characterized mainly by arrhythmia or weakness or sudden death. The means at his command could not have sufficed for a complete resolution of the problem. Yet even with the limited techniques then available, further progress could be made in the separation and definition of a class of persons suffering from functional heart disease. This was done, in soldiers of the Civil War, by J. M. Da Costa.

Case Reports

Successful Resuscitation from Cardiac Arrest Complicating Myocardial Infarction*

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WHILE CARDIAC MASSAGE is standard procedure for cardiac arrest in the operating room, it should become so in all cases of cardiac arrest due to myocardial infarction if the physican can arrive within five minutes of onset. As shown by this case, lives can be saved. Extensive review articles[1] on cardiovascular emergencies do not mention this procedure for such situations. Supportive measures of mechanical stimulation of the heart by forceful fist percussion on the precordium as recommended by Bellet[2] and good mouth-to-mouth artificial respiration allowed us to salvage a patient with no resulting damage to the brain. Krainer[3] states that the oxygen consumption of the brain can be reduced to 20 per cent of normal with electrical reactions being reduced to a minimum but that these can be fully restored after an unlimited revival time. Since the percentage of oxygen extraction from blood is greatly increased with slower passage, cerebral blood flow could be reduced well below this value. The advisability of withholding cardiac stimulatory and antifibrillatory drugs for a considerable period is again shown in this case.

CASE REPORT

The patient was a 51-year-old man with mild hypertension who had lost 30 pounds two years previously by dieting but had regained 10 pounds in recent months. Angina of five minutes' duration first occurred three days prior to admission and recurred for about fifteen minutes the day prior to admission. He had eaten a hearty supper but had not slept well. On awakening in the morning he had much gas and a constant substernal burning with an aching pain in the medial surfaces of both arms and in the teeth. The pain disappeared spontaneously in two hours but recurred a few hours later, lasting about one hour. He was entirely free of discomfort without the use of narcotics when transported to the hospital by ambulance.

Physical examination on admission on August 15, 1958 revealed moderate obesity and a blood pressure of 150/100 mm Hg. There was no cardiac enlargement to percussion, the rhythm was regular and the chest was clear. There were good pedal pulses and no pedal edema.

Cardiac Arrest and Treatment: Even though no pain was present, the patient was given Demerol® 50 mg intramuscularly on admission. One hour after admission a generalized convulsion developed. The nurse phoned one of us who ran up stairs to the patient who showed "black cyanosis" and took only two deep breaths. No pulse or heart sounds were present. Artificial respirations accompanied by forceful fist percussions to the precordium at a rate of 60 to 80 beats per minute were begun. A nurse went after a knife and found a surgeon who fortunately was close by. The chest was entered in the fourth interspace without entering the pericardium. The heart was in total arrest. Manual cardiac massage was begun. About fifteen minutes later, fibrillatory motions began and approximately five minutes later periodic ventricular contractions began so that manual systole was discontinued. During massage, mouth-to-mouth respirations were given with good respiratory excursions until endotracheal intubation was performed. The administration of intravenous sodium pentothal was necessary due to restlessness during the massage. No cardiac stimulatory or antifibrillatory drugs were used during the massage or in the early days after this. Maintenance of blood pressure required a concentration of 10 ampules of Levophed® (40 cc of 0.2 per cent solution) per liter of intravenous solution in order to restrict fluid intake to approximately 1000 cc plus the urinary output.

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Levophed was needed for 13 days. Demerol 25 mg. was given intravenously every fifteen minutes as larger doses caused respiratory depression and less frequent doses did not relieve marked restlessness.

Electrocardiograms: The electrocardiogram on August 15 showed sinus rhythm with rate 104, P-R interval 0.15, QRS duration 0.07, QT 0.32; ST depressed 1 mm in leads 1, 2, aVL, V_2, V_5, V_6; ST depressed 2 mm in V_3 and V_4; ST elevated 1 mm in 2, T slightly inverted terminally in 3.

On August 17, the electrocardiogram showed the P waves irregularly spaced with P-R varying from 0.08 to 0.16 second. The auricular rate averaged 70 and ventricular rate 100. This was probably basically sinus rhythm with many supraventricular beats from varying foci. There was an incomplete right bundle branch block pattern, with QRS duration 0.10 second. ST was elevated 1 mm in leads 2, 3, aVF, with T waves slightly inverted terminally. ST segments in other leads were less depressed.

The electrocardiogram on August 22 showed sinus rhythm, rate 102. P-R interval 0.13. Significant Q in lead 3. ST isoelectric and T waves coved and inverted in leads 2, 3 aVF, with T waves slightly negative in V_4 through V_6. QRS duration 0.08 second. The serial changes were those of acute posterolateral and inferior infarction.

Ten weeks after the infarction, quinidine, 1.2 Gm daily, was required to suppress ventricular extrasystoles. Roentgenograms indicate that the cardiac size was within normal limits. The patient is symptom-free.

Discussion

Bellet[2] in 1954 recommended that patients with cardiac arrest due to myocardial infarction should be given the benefit of cardiac massage. It was only because of employment of Bellet's recommendations that this patient's life was saved. Beck[5] and Reagan *et al.*[6] also have established the value of this procedure. Cardiac massage is considered standard procedure in the operating room when it complicates major surgery; there, however, the recovery rate is reported as about 50 per cent[7] to 60 per cent.[8] Under less ideal circumstances outside the operating room with poorer risk patients our salvage rate will probably not be as high. Actually any salvage rate at all would make the procedure worth while.

Prompt resuscitation prevented damage to the brain in this patient. While we were too busy to record time intervals, we are fairly certain that more than five minutes elapsed between the onset of cardiac arrest and the time at which massage was begun. During this period, good respiratory excursion was made by mouth-to-mouth insufflation. Some movement of this oxygenated blood to the brain was probably occurring due to myocardial contractions caused by very forceful fist percussions over the precordium at a rate of 60 to 80 beats per minute.

Massage through the unopened percardium as recommended by Ivory and Rinzler[4] seems preferred as it permits adequate massage. However, it lessens the likelihood of further arrhythmias and pericardial infections. Massive doses of antibiotics were used in our case and wound infection did not appear grossly.

As was shown by this case, cardiac massage permitted salvaging a patient when we were not really prepared for this procedure. We agree with Bellet[2] and Beck[5] that cardiac massage should be considered as standard procedure in patients with myocardial infarction with this complication.

Summary

(1) A case of cardiac arrest due to inferior myocardial infarction with successful resuscitation due to cardiac massage is presented.

(2) Forceful fist percussion to the precordium to cause cardiac contractions prior to opening the chest and artificial respiration effectively given by mouth-to-mouth insufflation contributed largely to the patient's recovery without residual damage to the brain.

(3) It is suggested that this treatment should now be considered as standard procedure in this type of case.

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Alternating Right and Left Bundle Branch Block Associated with Atrial Fibrillation and Complete A-V Block

An Unusual Case*

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THIS PATIENT presented a complicated conduction disturbance involving both the atria and the ventricles, with atrial fibrillation, left bundle branch block and complete atrioventricular (A-V) block. For two days the patient had the unusual electrocardiographic tracing of an alternating right bundle branch block (RBBB) and left bundle branch block (LBBB) with atrial fibrillation. The patient had episodes of Adams-Stokes syndrome and died during one of such episodes.

CASE HISTORY

The patient, a 57-year-old white male, was admitted to the hospital on December 13, 1957, for treatment of congestive heart failure due to hypertensive cardiovascular disease. He had the following previous electrocardiographic records taken in other hospitals during admissions for congestive heart failure:

1. 1950: Complete A-V block and RBBB (atrial rate 83, ventricular rate 38).
2. 1952: First-degree A-V block with LBBB (PR 0.31 sec, QRS 0.12 sec).
3. 1954: First-degree A-V block with episodes of second-degree (2 : 1) A-V block, LBBB, and premature ventricular contractions.
4. 1956: Same as in 1954.
5. 1957: February 25—second-degree A-V block with Wenckebach phenomenon and LBBB.
6. 1957: February 28—complete A-V block with LBBB.
7. 1957: November—first-degree A-V block with LBBB and premature ventricular contractions.

ECG Findings: The first electrocardiogram taken after admission showed atrial fibrillation with alternating RBBB and LBBB (Fig. 1). Another ECG taken one hour later showed atrial fibrillation with RBBB only (Fig. 2). The next several tracings taken the same day at 2–3 hour intervals showed again alternating RBBB and LBBB. On the third day the tracing changed to atrial fibrillation with LBBB only (Fig. 3). It remained unchanged for four days, when it changed again to atrial fibrillation with alternating RBBB and LBBB. Electrocardiograms repeated in the next four days showed again only LBBB with atrial fibrillation, but occasionally included complexes characteristic for RBBB. About five days later the patient developed a complete A-V block with LBBB and atrial fibrillation (Fig. 4). During that period, he had several episodes of Adams-Stokes syndrome and one of these episodes ended with heart standstill.

An autopsy demonstrated considerable hypertrophy of the right and left ventricles. There was also a recent anteroseptal myocardial infarction which was the cause of death.

DISCUSSION

Right and left bundle branch block are common conduction disturbances, due usually to an organic or functional block in one of the bundle branches.[1] The most common cause is damage to the bundles due to myocardial disease, as in arteriosclerotic heart disease and hypertensive cardiovascular disease. They are commonly associated with ventricular hypertrophy, and a left ventricular hypertrophy pattern may frequently precede the pattern of LBBB. The disappearance of Q waves may be the only sign of this change. There are some cases of LBBB with Q waves. They are explained by concomitant

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septal infarction[2] or by a block which is below the normal left-to-right septal conduction.[3]

Intermittent or transient bundle branch block is caused occasionally by toxic doses of quinidine or digitalis,[4] by peri-infarction block,[5] congestive heart failure, or chronic cor pulmonale. There are some cases, however, where no evidence of any cardiac disease was found,[6] and where the block can be explained by subclinical myocardial disease and in some rare cases by congenital abnormalities of the bundle branches.[6] Although extensive damage to the terminal network of any bundle branch may cause an electrocardiographic pattern of bundle branch block,[7] it is generally accepted that the latter is present when either one of the bundle branches is damaged or there is hypertrophy of a ventricle.

One case of an alternating right and left BBB in a patient with A-V block progressing from partial to complete was published by Cohn[8] in 1913. He explained it by alternative depression of conduction in the bundle branches. Katz, Hamburger, and Rubinfeld[9] have described a case with Wenckebach periods showing QRS complexes of RBBB or LBBB pattern with intermittent normal complexes, indicating normal intraventricular conduction. Another case reported by Miller and Perelman[10] showed LBBB when there was a complete A-V block, and RBBB when there was no A-V block. Some authors assume that there may be a difference in the refractory period of the two bundles which could account for the delay in one of the bundles.[11,12] In case of bilateral incomplete BBB

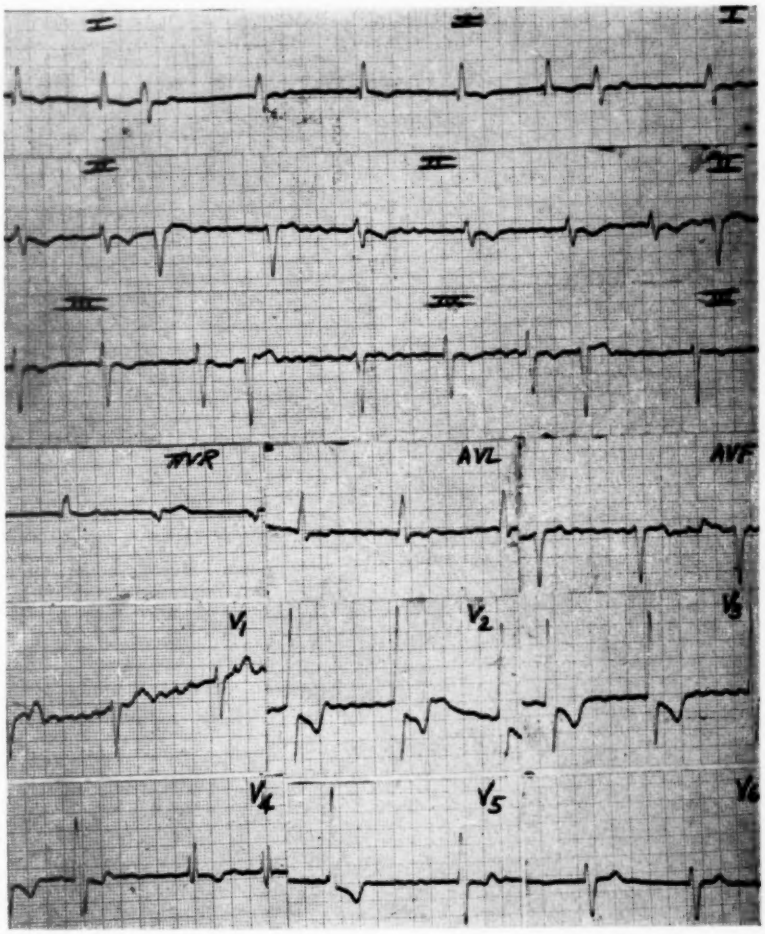

FIG. 1. Alternating right and left bundle branch block with atrial fibrillation. The first QRS complexes in L1, L2, and L3 are LBBB complexes. Compare with Figs. 2 and 3.

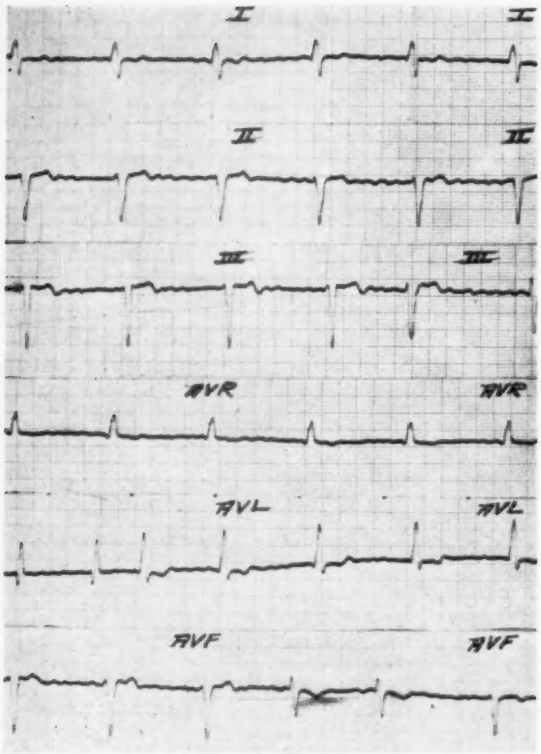

FIG. 2. Right bundle branch block with atrial fibrillation and complete A-V block. Last QRS complex in L3 is a LBBB complex preceded by an ectopic beat. The first and second QRS complex in aVL and the fourth and fifth in aVF are also LBBB complexes. The QRS intervals are equal indicating that there is a complete A-V block.

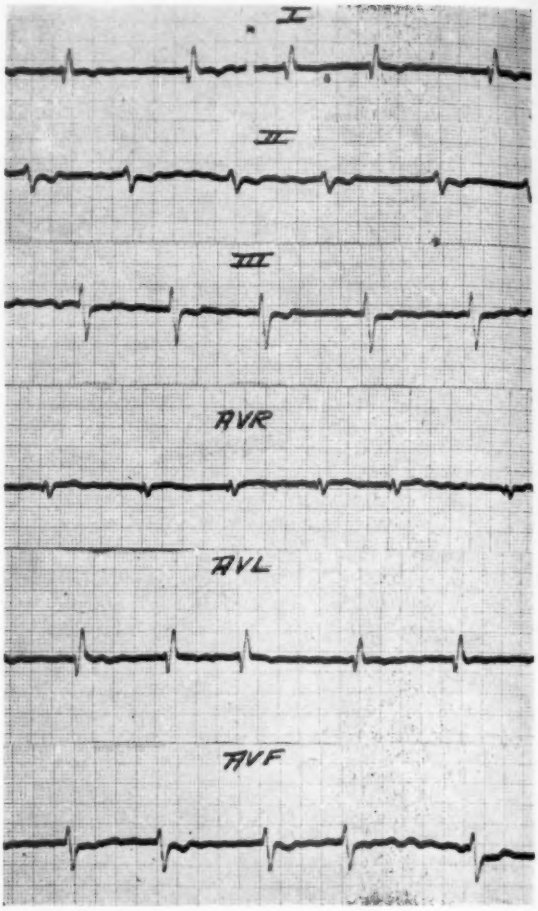

FIG. 3. Left bundle branch block with atrial fibrillation.

with equal delay there would be a simple A-V block without prolonged QRS complexes. Yater *et al.*[13] found that bilateral complete BBB may cause an electrocardiographic pattern of complete A-V block. Instances of complete A-V block with normal QRS complexes were explained by him by the presence of a pacemaker in the main stem below the block or by two pacemakers acting in both bundles simultaneously.

BBB and Atrial Fibrillation: The unusual feature in our case is the alternating right and left BBB associated with atrial fibrillation. Some cases of BBB in patients with atrial fibrillation can be explained by aberrant ventricular conduction if the supraventricular impulse reaches the ventricles before the refractory phase is over, but in these cases the QRS complexes are of different form and there are also normal QRS complexes. In other cases there was a BBB present before the patient developed atrial fibrillation, and therefore the QRS complexes showed the pattern of BBB. The cause of the block would be a lesion in one of the bundle branches or ventricular hypertrophy.

BBB and A-V Block: The QRS complexes characteristic for BBB would not change if a patient with BBB had in addition a first- or second-degree A-V block, as the impulses would be of supraventricular origin and the pathway of the impulses would remain the same. The situation changes completely when a patient with BBB has a complete A-V block. Here the pattern of the QRS complexes would depend upon the location of the pacemaker or pacemakers, if multiple, in regard to the lesion in the bundle branches. There are different possibilities as indicated in Figure 5.

There would be a pattern of LBBB if the lesion is in the left bundle branch and the pacemaker above the lesion (Fig. 5*A*), or a pattern of RBBB if the pacemaker is below the lesion in the left branch (Fig. 5*B*). There may be two alternat-

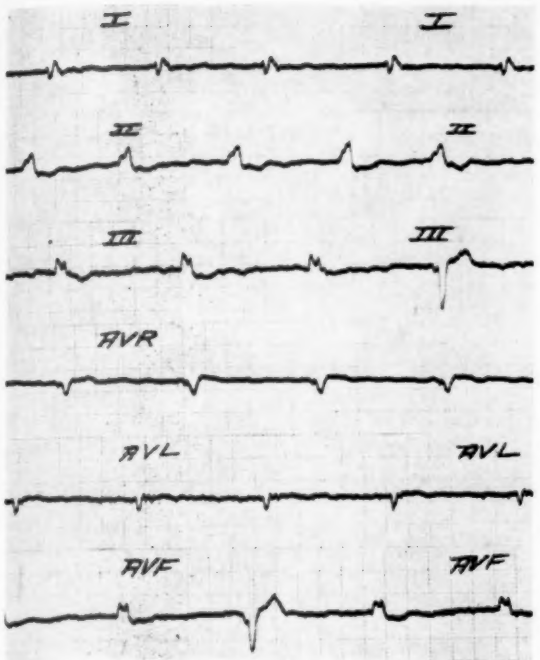

FIG. 4. Left bundle branch block with atrial fibrillation and complete A-V block. Last QRS complex in L3 and second in aVF are RBBB complexes. The slight difference in some QRS intervals is due to digitalis effect.

ing pacemakers, one above and the other below the lesion in the left branch; in this instance there will be an alternating right and left BBB (Fig. 5*E*). There may be cases of complete A-V block without any lesion in the bundle branches, showing patterns of BBB if the pacemaker is located in one of the branches (Fig. 5*C*), or alternating right and left BBB if there are two alternating pacemakers in each of the branches (Fig. 5*D*). We see that it is relatively easy to explain an alternating left and right BBB if we deal with a complete A-V block.

Causes of Alternating BBB in Reported Case: In our case, characterized by atrial fibrillation with slow irregular ventricular response and alternating right and left BBB, we have to accept as indisputable three facts: (1) the atrial fibrillation, occasionally changing to fast atrial flutter; (2) longstanding, permanent LBBB, due to a lesion in the left bundle branch; and (3) episodes of RBBB, which can be explained only by a pacemaker below the lesion in the left bundle branch.

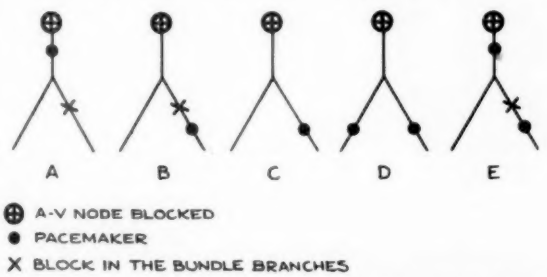

FIG. 5. A-V node and common bundle with both branches. Different locations of pacemakers and blocks with resulting conduction disturbances. *A*, LBBB; *B*, RBBB; *C*, RBBB; *D*, *E*, alternating LBBB and RBBB.

The slow irregular ventricular response can have three explanations: (1) Partial (first-degree) A-V block in a patient with atrial fibrillation may cause a slow irregular ventricular response, due to delayed A-V conduction of impulses from the atria. In second-degree A-V block we would expect prolonged pauses, corresponding to the dropped beats in a patient with regular sinus rhythm, which is not the case here. (2) The presence of an almost complete A-V block when, in addition to the idioventricular pacemaker, some impulses from the atria are conducted through the not completely blocked A-V node.[7] It occurs especially in cases with atrial fibrillation. (3) Complete A-V block with digitalis effect causing variations in the irritability of the idioventricular pacemaker,[7] also a frequent occurrence in cases of atrial fibrillation.

If we accept the first possibility (partial A-V block), the alternating right and left BBB can be explained only by alternative changing of the refractory period in each bundle branch, which would account for the delay in one or in the other branch.[11] This explanation is not satisfactory, as we would expect the irregularity of the ventricular response to be more pronounced; also the changing refractory period in the bundle branches is very improbable.

In case of the second possibility (almost complete A-V block), we would expect that only the impulses of supraventricular origins would be irregular, which is not the case here. The alternating left and right BBB could be explained as follows: The LBBB was due to supraventricular impulses conducted through the partially blocked A-V node, and the RBBB due to a pacemaker below the lesion in the left branch, acting only when the A-V block was complete (similar case described by Miller and Perelman[10]). Also, this explanation is not very satisfactory, as an alternating partial and complete A-V block with an idioventricular pacemaker acting only when complete block is present, although possible, is not very probable.

The third possibility, a complete A-V block with digitalis effect causing variations in the response of the idioventricular pacemakers, seems to be the most probable. In this case we should accept the presence of two pacemakers acting alternatingly, one below the lesion in the left bundle branch, the other above the lesion, as indicated in Figure 5*E*. It would explain the slow, irregular ventricular response, the alternating right and left BBB, the further development of a complete A-V block with LBBB (suppression of the pacemaker below the lesion in the left bundle branch), and episodes of Adams-Stokes syndrome.

Summary

Alternating right and left bundle branch block was observed in a patient with an eight-year history of conduction disturbance in the form of A-V block of all degrees and left bundle branch block. During his last admission, he had, in addition, atrial fibrillation and probably episodes of complete A-V block with Adams-Stokes syndrome.

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An Unusual Electrocardiographic Pattern Associated with Mild Myocardial Infarction*

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PATIENTS with clinically typical acute myocardial infarction not infrequently fail to present the usual electrocardiographic features associated with this condition. Among these we have observed cases showing a characteristic ECG pattern which to our knowledge has hitherto not been described, although undoubtedly observed by others. This pattern consists of *ST segment depression* associated with significant *lowering of the R wave*, usually in the same leads (Figs. 1 and 2). These findings were generally limited to a relatively small portion of the precordium, indicating that the infarcted area was not extensive. Mapping with an exploring precordial electrode showed the area producing this pattern to be generally small and surrounded by regions registering normal R waves and ST segments. In other cases, the diaphragmatic surface of the heart was involved and demonstrated this pattern in leads 2, 3, and aVF. A total of 13 cases have been observed.

Clinical Aspects: In some patients, the decrease in amplitude of the R wave occurred simultaneously with the depression of the ST segment. In others, it was not observed until several hours or even a day after the ST depression was noted.

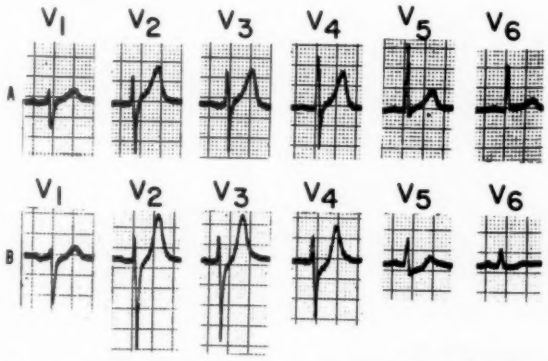

FIG. 1. Patient with acute myocardial infarction. (A) Control. (B) After infarction the next day, there is diminution in the height of the R wave in precordial leads associated with ST segment depression.

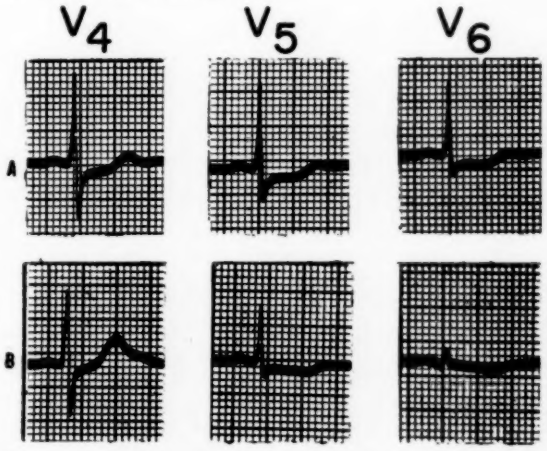

FIG. 2. Patient with acute myocardial infarction. (A) Immediately after infarction there is ST segment depression in V_4, V_5, and V_6. (B) The next day, in addition to ST depression, there is also marked diminution of the R wave in these l eads.

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The clinical course of all the patients showing this pattern was unusually mild. Shock did not occur. Congestive failure due to infarction was not observed. There were no fatalities. There was usually only slight evidence of tissue necrosis as determined by temperature curve, transaminase and sedimentation alterations. In most cases, the ECG eventually returned to normal.

Anatomic Basis of ECG Changes: A similar ECG pattern was observed experimentally in dogs some years ago. Histologically, the infarcts found responsible for this pattern in the dogs were not severe, and much viable myocardial tissue was present in the infarcted area.[1]

As shown in previous investigations, both the ST depression[2–4] and R wave lowering[5–9] result from changes in the outer layers of the myocardium (Figs. 3 and 4). While the deeper layers are undoubtedly involved in the infarction, they do not contribute to the electrocardiographic changes.

The clinical, electrocardiographic, and experimental evidence presented suggests that the infarcts responsible for this pattern are patchy and incomplete. Verification of this assumption must, of course, await histologic examination.

The ECG pattern described here should not be confused with another previously described pattern of myocardial infarction with ST depression. In the latter the course of the disease is much more severe, and death often occurs.[10,11] In such cases, the ST depressions are present over a much more extensive area of the precordium than the small region found involved in our cases.

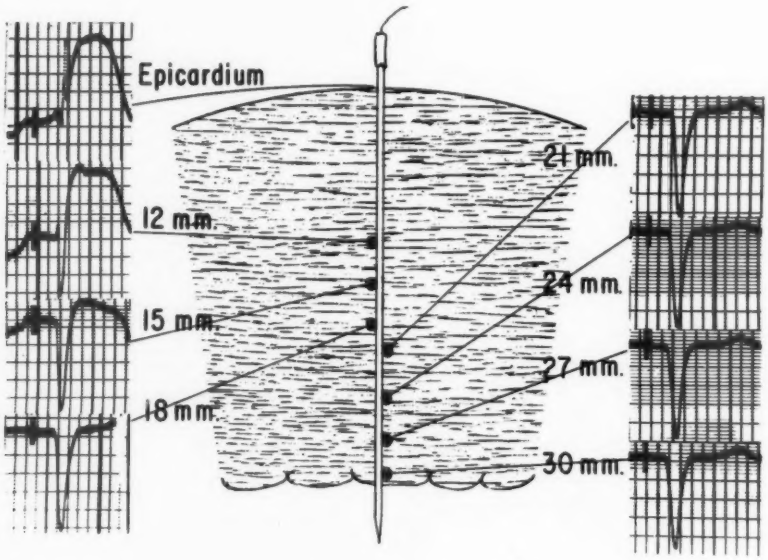

FIG. 3. Records obtained from various depths of the myocardium and from the surface. All the records taken from the inner half of the myocardial thickness are identical in every respect. The depolarization complexes obtained from the inner 15 mm of the myocardial thickness consist of pure QS waves. At 12 mm there is a small R wave and an S wave which is smaller than the S wave of the deeper layers. On the surface the complex shows no S wave. The ST segment is isoelectric in all records taken from the cavity to a distance of 18 mm from the epicardial surface. ST segment elevation makes its appearance at 15 mm below the surface and progressively increases in size as the electrode is moved toward the epicardial surface. (From Massumi, R. A., *et al.*: *Am. J. Med.* 19: 832, 1955.)

SUMMARY

(1) An unusual ECG pattern of mild acute myocardial infarction is described consisting of ST segment depressions and lowering of the R waves, usually in the same leads.

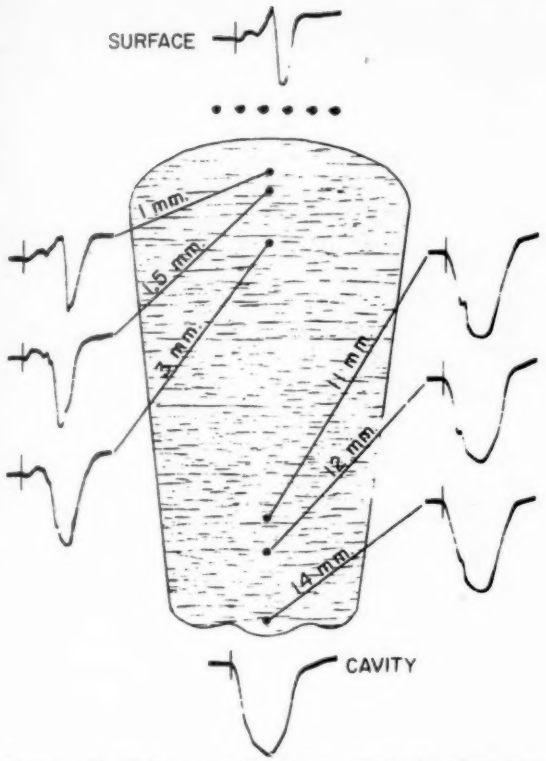

FIG. 4. Unipolar recordings from specified intramural levels of left ventricle in the dog. Common time reference. At subendocardial levels, a slur or embryonic R wave follows the intrinsic deflection. Little positivity is developed in these layers. In the outer layers, the R amplitude increases markedly. Note that the subepicardial complex closely resembles the surface complex, and the subendocardial complex closely resembles the cavity complex. Time marks recorded at rate of 120 per sec. (From Pipberger, H., *et al.: Am. Heart J.* 54: 511, 1957; reproduced courtesy C. V. Mosby Company.)

(2) The ECG abnormalities are usually limited to a small area of the heart.

(3) The clinical course of patients showing these changes is mild, and the ECG usually returns to normal.

(4) The infarcts responsible for this ECG pattern are probably patchy and incomplete.

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Readers are invited to submit reports of interesting cases and illustrative tracings for this department. These should not exceed 1,000 words in length. Although not necessarily original, all material submitted should have teaching value.

Progress Notes in Cardiology

Edited by EMANUEL GOLDBERGER, M.D., F.A.C.C.

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Use of Both Hypothermia and Extracorporeal Circulation for Open Heart Surgery

HYPOTHERMIA and extracorporeal circulation are complementary when used together for open heart surgery. Hypothermia reduces the body's need for oxygen, and therefore permits the use of a simple system of extracorporeal circulation. Ventricular fibrillation, the greatest theoretic hazard in hypothermia, is almost completely eliminated by the pump oxygenator; and, if it does occur, the arrhythmia can be easily reverted. The only remaining disadvantage of the combination is the cumbersome and uncertain technique of inducing and controlling hypothermia. This has been overcome by the introduction into the system of a simple efficient heat exchanger which permits easy induction of the hypothermia, certain control and rapid rewarming.

This method has now been used on 107 patients by Drs. Will C. Sealy, Ivan W. Brown, Jr. and W. Glenn Young, Jr. (Division of Thoracic Surgery, Duke University Medical Center, Durham, North Carolina). In 30 of the patients, external body cooling was used. In the remaining 77, hypothermia was obtained by a heat exchanger placed in the extracorporeal circuit. This latter device, which is very efficient, easily cleaned and reusable, has made it possible for them to reduce the body temperature to the desired levels of 28° to 30°C in from three to seven minutes and to rewarm the body to 35° to 37°C in a slightly longer period of time.

Oxygenation was obtained by plastic bags in the first 22 patients, but in the remainder of the series, this was discarded in favor of a modified DeWall bubble oxygenator. A finger occlusive type pump was used in all patients. In children flow rates of 50 cc per kg per minute have been employed, while in adults, rates of 20 to 35 cc per kg have been found to be ample. The maximum output of the extracorporeal system has not been allowed to exceed 2,400 cc per minute.

There has been, with this system, uniform lack of serious metabolic disturbances during a pumping period for as long as 90 minutes, as shown by the determinations of pH, pCO_2 and lactic acid. Of particular interest has been the high venous oxygen levels which have ranged from 57 to 88 per cent during the time of perfusion. In none of the group has any damage to the central nervous system developed that could be attributed to the extracorporeal system.

The mortality for this entire series is less than 18 per cent. There have been no deaths in a group of 50 patients with atrial septal defects and none in the group of patients with pulmonic stenosis. In the patients with ventricular septal defect and tetralogy of Fallot, there has been a mortality rate of 14 per cent, but there have been no deaths in the last 30 patients. Since the use of the heat exchanger, the mortality figure is less than 7 per cent.

In all patients undergoing ventriculotomy the heart was arrested by the injection of potassium citrate (0.81 per cent), magnesium sulfate (hydrated 2.47 per cent), and Prostigmine® (0.001 per cent) solution into the coronary arteries. The heart readily resumed sinus rhythm in nearly all patients; and if ventricular fibrillation occurred, it was easily reverted with an electric shock. Both the experimental studies and the clinical experiences confirmed the fact that the cold heart is much more tolerant of coronary artery occlusion than the warm heart.

This method offers many other advantages such as protection in case of pump-oxygenator

failure, rapid induction and exact control of hypothermia, and simplicity in application. Lastly, it offers the possibility of utilizing hypothermia at still lower levels; so that prolonged periods of low perfusion rates can be accomplished without the associated trauma to the blood and other cumulative effects that occur when the high flows that are demanded by normal body temperatures are used. In view of this, the Duke University group has now studied patients whose temperature has been reduced to 21°C and has made laboratory studies with temperatures below 10°C.

Hypocalcemic Electrocardiographic Abnormalities in Patients With Liver Disease

An interesting electrocardiographic study of cirrhotic patients has recently been completed by Dr. David A. Spodick (The ECG Laboratory, Lemuel Shattuck Hospital, Boston). Electrocardiograms resembling those in hypocalcemia (QTc prolongation due to independent RS-T elongation) were found in 18 of 194 patients with liver disease. There was no relation to serum calcium, potassium, or other biochemical findings. In most patients, calcium was normal or slightly elevated, potassium normal or slightly depressed, and bilirubin normal or slightly elevated. Five cirrhotic patients with this abnormality have been given 20-minute intravenous infusions containing 450 mg calcium given as 50 cc of 10 per cent calcium gluconate-glucoheptonate (Abbott), with diminution of the QTc and gross decrease of the RS-T segments. The identical procedure in four other cirrhotic patients without abnormal RS-T segments resulted in some QTc diminution but no RS-T shortening.

Electrocardiograms resembling those in hypocalcemia have been obtained in patients with liver disease and in experimental injection of bile constituents, some of which can actually depress serum calcium, possibly by means of chelation. Hyperestrogenemia (which may occur in parenchymal liver disease) can greatly alter the binding of serum calcium.

While the meaning of these findings is not clear, there may be a decrease in the metabolic availability of the circulating calcium. Further studies are in progress. Collateral studies on the relation of these phenomena and the administration of intravenous calcium and their effect on sodium turnover in the cirrhotic subject are being completed by Dr. Spodick in collaboration with Drs. Robert S. Morrison and Alberto Mazzoleni.

Cardiac Resuscitation

Edited by PALUEL J. FLAGG, M.D., F.A.C.C.*
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The Heart, A Hemodynamic Pump

CARDIAC resuscitation from standstill or fibrillation requires immediate reestablishment of contraction, rate and rhythm. Manual compression must provide enough systolic pressure to sustain cerebral and coronary pressures to prevent hypoxia with ensuing decerebration, and to restore the integrity of the myocardium. Forceful and rhythmic compression of the heart is not enough. At the end of compression, pressure must be released completely to permit the chambers of the heart to refill.

Cardiac resuscitation must be a conditioned reflex. When emergency resuscitation is necessary, there is no time for differential diagnosis between vagal arrest and myocardial failure, or for the trial and error of external defibrillation. The sequence of action should be worked out and practiced before the accident. If unprepared to respond promptly with correct reflex action when faced with such an emergency, we call for help and the patient, who might have been saved, dies.

Full alveolar oxygenation before cardiac resuscitation is mandatory. Air or oxygen must be blown into the trachea. Transpharyngeal insufflation (blowing air across the pharynx by mask or pharyngeal tube) is a half measure which has often invited unnecessary failure. A relaxed airway is easy to expose. Anyone can intubate the airway with practice. Are the time and effort necessary worthwhile? Definitely yes! It is not out of order to devote a few hours in becoming acquainted with the structures of the airway to save a life. Facilities for such instruction are available and should be greatly multiplied.

THE REGULATION OF THE HEART BEAT

Granted the hope of success, which rests upon oxygen in the blood to be circulated, a brief review of what induces the heart to beat is in order. The properties of the myocardium include: excitability and contractility, rhythmicity, and conductivity.

EXCITABILITY AND CONTRACTILITY

Excitability is the ability of a tissue to respond to a stimulus. In cardiac muscle this causes a shortening of the fibers termed contraction. Certain features of the contraction of cardiac muscle require consideration.

The "All or None" Law: This law states that the weakest or minimal stimulus causing contraction will produce the maximal contraction. When it is said that the cardiac muscle follows the "all or none" law, this applies only under the conditions existing at the time. The excitability and contractility of the muscle is variable and influenced by the length of the fiber as expressed by Starling: "The energy set free at each contraction of the heart is a simple function of the length of the fibers composing its muscular walls." In other words, the greater the venous return, which stretches the heart muscle fibers, the greater will be the contraction and so the cardiac output. This is the "Law of the Heart."

Hydrogen Ion Concentration: Acids favor relaxation and depress conductivity of the cardiac muscle. The heart rests in diastole. *Alkalis* prolong systole and shorten diastole. They increase conductivity through the auriculoventricular bundle. When the pH of fluids perfusing the heart is reduced, complete heart block occurs.

During muscular exercise, the higher tension of carbon dioxide probably benefits cardiac behavior. During exercise, the blood flow through the muscles is greatly augmented and a larger volume of blood is returned to the right side of

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the heart. The rise in carbon dioxide tension favors more complete relaxation of cardiac muscle to accommodate the greater load of venous blood and Starling's Law comes into play.

Cations: About 1880, Ringer observed that if the heart were perfused with an osmotically adequate solution of *sodium chloride*, a few beats occurred, but the heart then *stopped in diastole.* The addition of *calcium* restored the beat for some time but the heart again came to a standstill, this time in *systole.* The *addition of potassium* not only antagonized the calcium effect, but started and maintained the heart beat.

In the absence of potassium, calcium in excess or in normal concentration will lengthen systole at the expense of diastole. The heart finally stops in the fully contracted state (calcium rigor).

Potassium acts reversely if in excess or unbalanced by calcium. Diastole occupies more of the cardiac cycle and the heart ultimately comes to rest in a completely relaxed state (potassium inhibition). A solution of calcium and potassium alone will not sustain the beat. Sodium is essential, but the manner in which it acts is not clearly established.

Little is definitely known of the underlying physical or chemical changes through which these elements influence the heart beat. Electrophysiologic evidence points strongly in the direction of permeability changes of the cell membrane, setting up differences in electrical potential on the two sides of the membrane.

Other factors influencing excitability and contractility are fatigue, nutrition and disease, temperature and oxygen supply.

RHYTHMICITY AND CONDUCTIVITY

Rhythmicity of the heart after removal from the body can be maintained for hours by perfusing the coronary arteries with a suitable solution. The factors discussed in the previous section naturally influence rhythmicity and conductivity too. There is a close integration between the three or four basic properties of the heart muscle. This division is unnatural but convenient and helpful for remembering.

Neurogenic Control of the Heart

All the nervous and muscular connections between auricles and ventricles can be severed in the mammalian heart (Stannius ligatures) without stopping the ventricular beat which, after a pause, will reappear at a rhythm independent and slower than that of the auricles. A descending scale of rhythmicity has been shown in this order: S-A node, A-V node, A-V bundle, the bundle branches and the Purkinje system. The highest rate of rhythmicity is located in the S-A node, called the "pacemaker" of the heart. From here electrical discharges start, spread over the auricles, and reach the A-V node, where conduction is slowed slightly. The impulse then spreads by way of the aforementioned pathways to the ventricles.

Rhythmicity, conductivity, excitability and contractility of the heart are integrated and adapted to the general needs of the body by the central nervous system. In the intact animal or human being the automatic action is under the continuous influence of nervous impulses arising from the cardiac centers in the medulla. From these centers the vagus and sympathetic systems transmit impulses to the heart. Afferent pathways from the heart travel in the right vagus and in the aortic nerve. Other stimuli arise from numerous regions of the body. The present discussion is limited to the effect of the vagus and sympathetic nerves.

Action of the Vagus: There are probably two types of vagal fibers, one type producing a change in cardiac rate (*chronotropic effect*), the other depressing conduction (*dromotropic effect*). In mammals the vagus nerves affect the heart by action on the auricular muscle and the junctional tissues. They exert no direct action on the ventricular muscle. All effect on the ventricles are secondary. Carotid sinus pressure is thus useful in auricular tachycardia but not in ventricular tachycardia.

If the peripheral end of the cut vagus in a dog is stimulated, diastole is lengthened, the heart slows or stops completely, and the blood pressure falls toward zero. When complete stoppage of the heart is caused by vagal excitation, the ventricles, but usually not the auricles, resume beating after a time although the stimulation of the nerve continues. This is "*vagal escape.*" The ventricles begin to beat at their own rate of about 40 to 50 per minute.

Vagal effects occur from a variety of visceral stimulations—traction on the mesentery, irritation of the carina during intubation, and carotid sinus pressure. Hypoxia sensitizes the myocardium to the arresting effects of vagal stimulation and may predispose to ventricular fibrillation. In practice, this is seen in the accident of arrest

in the anesthetized, hypoxic person being intubated, since a well oxygenated myocardium is not affected by vagal stimulation. This accident is not reported in the conscious, well oxygenated patient who is undergoing bronchoscopy.

Action of the Sympathetic (Accelerator, Augmentor) Nerves: Stimulation of the sympathetic nerves increases the heart rate of auricles and ventricles, and the force of contraction. The sympathetic nerves exert a direct action on the ventricular muscle. Stimulation of the accelerator nerves may excite the ventricles and induce fibrillation. Fibrillation of the auricles, on the other hand, is not induced by this means.

Myocardial Hypoxia, Cardiac Arrest and Fibrillation

Gases and drugs also influence the heart beat. Perhaps the most important factor is the lack of oxygen which first increases the rate and reduces the output. The heart then dilates from its own venous congestion and from systemic congestion in the large veins. As the heart dilates, the muscles are stretched, increasing the contractility of each fiber. As hypoxia increases the beat gradually slows to complete arrest or the myocardium is sensitized to vagal stimulation. The latter leads to cardiac irregularity and then fibrillation.

Changes in rate, blood pressure and cardiac output may result from a reduced lumen of the coronary vessels (70 per cent or more), or by spasm of the coronary arterioles, shutting down the supply of blood in the capillary bed.

Fibrillation does not occur as the direct, uncomplicated effect of hypoxia. Where segments of the heart muscle are hypoxic through coronary occlusion, the blood electrolytes effect a varying potential between the affected segments and the normal myocardium, to all intents electrocuting the heart as effectively as a low amperage house current. Of all deaths from heart disease 85 to 90 per cent are said to be caused by ventricular fibrillation. Prompt massage of a fibrillating heart will sustain blood pressure and protect the brain, if adequate respiratory resuscitation has been previously given or is simultaneously applied. Such a heart cannot be defibrillated by massage or a defibrillator, unless the myocardium is oxygenated. Hence, external defibrillation after a minute or two of complete arrest is futile.

Cardiac Massage: Cardiac surgery has revealed and made familiar through manual manipulation the "feel" of the heart as a tough, resilient, automatic entity. Given the essential support of oxygen and the mechanical assistance of massage, the heart can be expected to respond from arrest by simple massage; from fibrillation by massage and mechanical defibrillation.

As one reflects upon the intrinsic competency of the heart to function if provided with oxygenated blood, Beck's often repeated statement, "This heart was just too good to die" is easily understood. All it needs is help during a sudden, intrinsic, electrical execution or arrest from an unrecognized progressive hypoxia. Seemingly drastic surgical intervention loses its repugnance when a life-saving purpose exists. Intelligent massage, in the manner to be described in another article, becomes an immediate, non-traumatic support of the blood pressure until normal beats are resumed or deliberately induced by a defibrillator. But do not delay, when indicated do it immediately and you will save a life!

Workmen's Compensation for the Cardiac

Relationship of Strain to an Acute Cardiac Insult

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IN a previous communication[1] I presented a short outline of a study made in New York State under the direction of the Moreland Commissioner,† Judge Callahan, relating to certain compensation problems. The medical phases of the study were intended to determine the possible relationship of strain to cardiac disability. It consisted of a questionnaire sent out to a number of cardiologists and internists with reference to the problem, and the answers received. The first question dealing with the possible relationship of work to heart disease has been discussed by me in another communication.[2] In the present paper, we shall take up the second question which reads as follows:

"Suppose the case is of a 60-year-old workman employed for 20 years in a job that regularly required the lifting of 100 pound weights. During the course of this work as customarily performed, a coronary occlusion with myocardial infarction develops, while lifting a 100 pound weight. From a medical viewpoint, would you consider the attack to be causally related to the lifting of the weight?"

Of 383 replies received, 185 or 48.4 per cent said yes or yes with some modifications and 197 or 51.6 per cent said no. Of those who replied in the affirmative, with modifications, a few believed that there was a "possible" relationship, or a "contributing," "precipitating," or "aggravating" factor that acted as a "trigger mechanism" and a few that infarction could occur as a result of coronary insufficiency without coronary occlusion.

Those who replied in the negative, based their views on the assumed facts that the man was doing his customary work to which he was conditioned; that there was no unusual effort or strain; or, that the heart attack was imminent and it was merely a coincidence that it occurred while he was working.

VIEWS OF THE COMMITTEE

I submitted this question to our Committee members and the opinions expressed by the men who answered were as follows:

One member replied: "I think the myocardial infarction was only coincidental since this man was doing his customary work. However, if this man's occupation had been a clerk which required no physical exertion and he had had a myocardial infarction while shoveling snow, it would be my opinion that there was a causal relationship."

Another member, in his answer to the first question which, in the nature of his reply, is applicable also to the present question, wrote in part as follows: "I have seen a number of patients who have had heart attacks while performing duties that were unusually strenuous and not in their usual line of work. It has always been questionable in my mind as to whether or not the exertion was a precipitating factor or merely a contributing factor. Certainly, in my experience, more patients seem to suffer their attacks while not working than while working.

It would seem to me that if the answer to the question asked was answered in the affirmative, no employer would ever be willing to hire a person in an age in which most heart attacks occur. Also, if the employer were to be responsible for such attacks it would seem to be prudent that he fire all employees above

* Chairman, Workmen's Compensation Committee, American College of Cardiology.

† It has been brought to our attention that this survey was made by the Medical Consultant appointed for the investigation and had nothing to do with the Workmen's Compensation Board itself. Neither the questions nor the conclusions reached were passed upon or published by the Board.

such age and replace them with younger men. Certainly, if the employer is held responsible it will be increasingly difficult for anyone 40 years or older to either obtain or retain employment."

A third member replied the following to the first question which also applies to the second: "It seems to me that many of the differences of opinion that are rendered by physicians on this subject are due to the inherent ambiguity in the law. As I understand it, if something probably has played a part in the production of the disability and this something is connected with a person's work, then the illness is compensable. If this is only a possibility, then it is not compensable. It is my understanding that, in general, psychosomatic illnesses are ordinarily not judged compensable. For example, it would be difficult to argue that an extremely unpleasant workman's situation with a good deal of tension and strain is not responsible for the production of a duodenal ulcer and as such, a duodenal ulcer that develops under these circumstances perhaps should be considered a compensable injury. However, such has never been the case or certainly not very frequently.

If such reasoning should ever be established as a cause of compensable injuries, then there hardly are any limits to what compensation would and could cover and in effect, compensation would become health insurance for the worker. I believe this is fundamentally wrong. I do not believe that this was ever the intent of the law, and I think that it is terribly important to establish certain principles so that such illnesses will not be adjudged compensable in the future.

If one wants to reason that such factors may play a part in the development of arteriosclerotic heart disease and myocardial infarction, I shall have to agree that it is true and I think most physicians would agree. However, it would seem to me to be a terrible mistake to ever permit such illnesses to be adjudged compensable."

Dr. I. Robert Frank of Boston, Massachusetts, in answer to the first question gave a lengthy and interesting discussion on the subject which may apply also to the second question presented in this paper. The following are pertinent parts of his expressions: "I frankly believe as do most observers in this field, that exertion, as such, cannot cause or produce heart disease. In some cases, it may aggravate a previously damaged heart. . . . In general we all recognize that coronary sclerosis is the common denominator in all but very few cases of coronary arterial disease. By virtue of its presence, coronary thrombosis occurs, to be followed in most cases by coronary occlusion and myocardial infarction when, and if, the time and situation arises. . . . Physical strain may increase aortic pressure and intracoronary pressure transiently, producing acute changes in coronary vessels where marked atheromas might exist; or emotionalism and strain may stimulate the sympathetic nervous system, thus increasing the epinephrine output leading to vasoconstriction and increase in blood pressure, as well as to tachycardia, with resulting cardiac hyperactivity and increased demands upon the coronary blood supply; or that intimal hemorrhages brought on by strain. . . may permit blood to seep into the intimal lining, thus lifting atheromatous plaques into the lumen, causing occlusion, and possibly even by disruption and dissolution of intima lead to liberation of thromboplastin, which, of course, is one of the factors responsible for the clotting mechanism of blood. . . I believe that all these are in the background of answers to the question itself.

In answering the question itself, I would like to make a few observations. . . . In the first place, a man who has been working at a certain job for many years has aged many years. . . .Atherosclerosis will probably be present in his coronary arteries even if it was not present twenty years before. Thus, he could be more vulnerable to an attack of coronary occlusion with much less stimulation, than 20 years before. However, this hypothetical attack is much more apt to occur while he is sleeping in bed, resting, watching an exciting program on TV, playing cards, etc., than while actually working. This is proved by statistics. . . .In the second place, emotionalism, in my opinion, does enter the picture very often. A man may have. . . an argument . . . or after a party the night before which ended about 2 A.M. he comes to work, and about two or so hours after performing the same duties that he has had for many years he has a coronary occlusion, suddenly without any warning. It is obvious that an extra employment factor has entered the picture. . . . In the third place, I believe that the presence of other constitutional diseases. . . enters the picture, inasmuch as such people are more apt to be subject to coronary problems than people in excellent physical condition. .

It would be my opinion, therefore, . . . that our question cannot be resolved into an affirmative or negative conclusion as such. It is my impression, however, that. . . chances of getting a coronary attack from the work alone, are quite remote, unless. . . other factors enter the picture, particularly emotionalism. . . ."

I fully agree with the essence of the statements made by my colleagues. Let us attempt, however, to analyze the situation and the facts more fully. In doing so I shall offer some of my personal views which are, of course, subject to refutation and possible modification at a later time.

The Socioeconomic Problem

We are all fully cognizant of the fact that compensation payments made for cardiac disabilities, especially in the arteriosclerotic group which comprises the greatest number of cases, has a double-edged effect. It burdens

the defendant with a tremendous overhead and it carries the danger of depriving the older workers of employment. These problems, however, are not and should not be solved by the physician. They are problems for the legislators, economists, and philanthropists. Our testimony must in no way be affected by them. We must realize that when a man begins to work at about 40 years of age at a menial occupation and continues for a period of twenty years, the possibilities of accumulating enough money to carry him through a long period of illness or a disability which may continue the rest of his life, are slim. The salary he earned is not enough to help him save for a calamatous illness. If he is struck by myocardial infarction at the end of twenty years, some provision must be made in the law for his protection. If there is some causal relationship between the work he had performed at the time of the accident and the myocardial infarction, he should be compensated by the employer regardless of the financial strain on the employer and of the effects on the worker's future employment. For this reason, the law in most states takes a very liberal view in these cases. It considers a case compensable even if the strain which caused the attack is not the sole cause or even the main cause, provided it is of sufficient severity to help bring about the attack.

We, as doctors, have no legal right to look at the case from any other than a medical viewpoint. Our sole duty is to give honest and logical testimony in order to help solve the often knotty medical problems involved. If the facts in the case point to causal relationship, even if it is not the sole cause, the plaintiff is entitled to protection by appropriate testimony. If there is no possible causal relationship, the defendant should be protected. It is also the legal and moral duty of the doctor to help eliminate dishonest claims and malingerers and to help in rehabilitating the claimant so that he may return again to a productive life. It is only along these channels that we, as doctors, can be of help in solving the socioeconomic problems involved in these cases.

The Medical Considerations

Although full knowledge is lacking as to the pathogenesis of coronary atherosclerosis and of an acute coronary insufficiency, occlusion, and the resulting myocardial involvement, enough is known or can be inferred from the pathologic findings observed at autopsy and correlated with the clinical history to form a reasonable opinion.

In order to obtain a comprehensive picture of the situation we, as doctors, must learn to look at pathogenesis from a broad viewpoint. All possible factors that help to bring about a breakdown in any structure or organ of the body must be taken into consideration. We must not pinpoint one factor as the only cause of such breakdown. Even in known infectious diseases, such as tuberculosis, for instance, it is not the tubercle bacillus alone which is responsible for the disease. There are many additional factors, such as lowering the resistance by overwork, improper rest, poor food, crowded environment, insufficient fresh air and sunshine and, most important of all, individual susceptibility, which are contributory. In conditions such as coronary disease, the theory of a multiplicity of underlying causes is certainly to be entertained.

That coronary atherosclerosis is a slowly developing progressive process over many years has been discussed before.[2] It has also been pointed out that "aging" is not a factor in its pathogenesis. In the early stages it appears to be initiated by certain physiologic processes. The progress of the disease is due to a continuation of these processes associated with certain local metabolic and chemical changes and cellular alterations of an irritative nature. These progressive processes must ultimately come to a stage when an acute process develops in a degenerated area of the coronary arteries, at which time the clinical manifestations of the disease begin to exhibit themselves.

The Acute Phases of Coronary Disease

The acute phases of coronary disease may consist of one or more of the changes well enumerated by Dr. Frank in his discussion in this paper. These alterations may result in functional intermittent myocardial ischemia alone or more prolonged ischemia, ischemic necrosis, and infarction. These are the underlying causes of the clinical manifestations of coronary disease. Before these changes occur, coronary disease is asymptomatic.

Intermittent myocardial ischemia begins to appear when the coronary atherosclerotic process has reached a stage which interferes to a greater or lesser extent with the blood supply to the myocardium. It exhibits itself in the an-

ginal syndrome. Freedberg and co-workers[3] observed that in nearly all their autopsy cases, patients who suffered from angina during life showed old complete occlusive processes in at least one main coronary artery. From clinical experience, we all know that the anginal syndrome may last at times many months or years. We also know that at times many days, months, or even years may pass by without any disturbances after the patient has had anginal symptoms for weeks or months. This may be explained on the basis of the development of a collateral circulation, which Wiggers[4] has shown to occur in slowly developing coronary narrowing. Furthermore, we also know that the attacks may be brought about by extracardiac factors such as excitement, anxieties, cold weather, overdistention of the stomach by food, postprandial lipemia reaching a peak several hours after the ingestion of food,[5] pathologic conditions in organs above and below the diaphragm which may act as trigger mechanisms, and other conditions. Most of these are explainable on the basis of reflex coronary spasm and at times on an increase in adrenalin secretion and metabolic activities. All of these would speak for the fact that besides the coronary disease, extrinsic factors enter into the picture.

Physical strain, which increases the cardiac work, is the most important factor in bringing about attacks as is well known to all of us. Even here it is not the physical work alone but associated neurogenic, reflex, and other factors mentioned herein which enter as causes, evidenced by the fact that a person can do a considerable amount of physical work at one time and not at another, under different conditions. Thus, a person may be able to do much walking in his place of business, but may suffer pain when he does the same amount of walking on the street. Also, the pain may be absent when he walks a certain time of the day and be severe at other times, especially after a meal. The emotional element is also an important factor in bringing about an attack, as is well known from clinical experience.

It is thus seen that even at this stage of coronary disease there are many factors which cooperate in bringing about the anginal syndrome, although physical activity is the predominant one. Remove this factor and the patient is freed from the attack in most cases, especially in the early stages.

When acute structural alterations in a coronary vessel or vessels are more severe, the symptomatology becomes more marked and prolonged, its severity and prolongation depending a great deal upon the degree of structural myocardial changes. These changes may vary from mere prolonged ischemia to focal necrosis and gross severe myocardial infarction, depending mainly upon the degree of interference with the blood supply to a given portion of the heart caused by the acute coronary process. It is important to remember that marked myocardial changes may occur without complete occlusion of a coronary vessel and relatively little or even no damage may occur in a complete occlusion. It depends partly upon the extent of the collateral circulation but mainly to some of the other extrinsic factors already mentioned, especially the neurogenic and reflex elements. In Yater's series,[6] of 950 autopsies performed on patients who died of coronary disease, 13 per cent had no coronary occlusion in the presence of myocardial infarction and others had no infarction in the presence of occlusion. They also found that occlusion, when present, was due to atherosclerosis alone in 39 per cent of the cases, to thrombosis alone in 23 per cent and to combined sclerosis and thrombosis in 25 per cent.

Physical effort, especially when associated with emotional disturbances, may be a precipitating cause of an acute coronary and myocardial insult of any form mentioned herein. That the latter much more frequently occurs apparently "spontaneously" at rest or while sleeping, does not mean that there are no causative factors in bringing on the attack in an individual whose atherosclerotic process is "ripe" for such attack. Thus, if the attack occurs while at rest in bed, it may conceivably be due to slowing of the circulation, thus predisposing to thrombosis. It may also be due to postprandial lipemia after ingestion of a heavy meal in the evening. It may be due to emotional strain under which the sufferer has been working during the day and which deprived him from proper sleep and rest during the night or predisposed him to certain frightening dreams. Nervous tension and strain during work in general, appear to have, in some cases, a long-term effect in predisposing a person to acute coronary changes. Friedman and co-workers[7] observed changes in the serum cholesterol level and blood clotting time in

accountants during the time of the year when they were compelled to work under severe strain to meet a deadline. It may also be due to a gradually mounting tension of an emotional origin of months' or years' duration, which Weiss and co-workers have observed to occur with great frequency in some cases of coronary occlusion. We can thus see that there is undoubtedly always a cause for an acute attack, although we may not immediately discern it. I doubt if there is such a thing as "spontaneity" in biologic processes.

Application to Our Case in Question

Considering the case presented herein for our opinion, we will observe from the discussions that the question is not specific enough and leaves out some elements that we would want to know before answering it. (1) Under what environmental conditions was the man working at the time of his attack? (2) Did he have any disturbing emotional experiences in recent hours? (3) How fast and how high did he lift his 100 pound load at this particular performance? (4) Did he work at this time after a heavy meal or after a sleepless and restless night or other adverse conditions which would lower his resistance? These and other factors must be taken into consideration in answering the question.

I believe, however, that whatever other factors might have been present which predisposed this individual to the attack, an important one of which was the advanced state of the atheromatosis, the most important immediate cause was the lifting of the load. This was "the last straw that broke the camel's back." It was the final factor which resulted in the attack and, as such, it should be considered causally related. The chances are that had he avoided this type of work at a period when he was predisposed to an acute attack by the various extrinsic factors, the attack would not have occurred. Therefore, even though the lifting of the load could not be considered the sole cause, it is certainly the important cause.

It is interesting to observe that most clinicians who believe there is no causal relationship between work of the kind this person performed and his attack, would hesitate in allowing a 60-year-old man to lift a 100 pound weight. Evidently, they believe that such work at this age is hazardous even though the worker has been doing it for many years.

Summary

The problem of causal relationship between physical strain of lifting a 100 pound load, which a worker was accustomed to doing for 20 years, and a resulting myocardial infarction is considered in this paper. It is pointed out that myocardial infarction or other myocardial damage due to coronary disease is caused by acute structural changes in a coronary artery or arteries. There are many underlying causes which participate in precipitating the acute structural changes, such as lowering the resistance of the individual by insufficient rest and sleep, emotional disturbances, anxieties, extrinsic and intrinsic reflex factors, the advanced state of the atheromatous process, and others.

It is believed that under such circumstances lifting of a heavy load may bring about an attack even if the same type of work was performed before without any apparent ill effects, and is therefore to be considered causally related. All acute disease processes are known to have a multiplicity of predisposing factors but the predominant cause in this case is the lifting of the load, which is to be considered the main responsible element.

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Announcement of a Study on the Legal Principles Involved in Cardiac Disability Due to Strain and Trauma

The differences in the treatment of claims involving heart attacks by courts and workmen's compensation commissions is the subject of a new study being made under a recent grant by the National Heart Institute of the U. S. Public Health Service. Associate Dean Harold F. McNiece, of St. John's University School of Law, has been named director of the study.

When a workmen's compensation award is made to a victim of a heart attack, it is often based on a finding that the stress and strain of the work being performed caused the attack, or that the attack was an "accident" arising out of and in the course of employment. When an award is denied, it is usually on the assumption that no "accident" occurred, or that there was no causal connection between the work and the attack. However, the variations of opinions in different states range from the view that almost any on-the-job heart attack is compensable to the opposite extreme. As a result, the legal status of the victim of a heart attack has become uncertain.

The McNiece study group plans to explore in detail the legal principles which are being applied and the procedures being utilized by courts and workmen's compensation commissions when granting or denying awards to persons who suffer coronary attacks while at work. Thus, they hope to develop information that will lead to more equitable and consistent handling of these cases.

The investigation will focus particular attention on how courts and commissions approach the question of whether or not heart attacks were caused by stress, and will try to determine to what extent medical testimony is heeded. The effect of testimony by impartial medical experts will also be explored.

Others associated with the investigation are Paul E. Gurske, former Director of the Bureau of Labor Standards of the U. S. Department of Labor, who is to assist in the statistical aspects of the study, and a number of experts in the medicolegal field who will act as consultants, including Professor L. Whiting Farinhold of the University of Maryland School of Law, past chairman of the medicolegal committee of the Association of American Law Schools; Barnett S. Fox, former New York State workmen's compensation referee; Dr. Milton Helpern, Chief Medical Examiner of the City of New York; Dr. Irvin Klein, Medical Director of the New York State Workmen's Compensation Board; Professor John V. Thornton of New York University School of Law, and Theodore C. Waters, of the Baltimore Bar.

Anyone who might have any materials, suggestions, or views to offer is invited to get in touch with Associate Dean McNiece at St. John's University School of Law, 96 Schermerhorn Street, Brooklyn 1, New York.

Minutes of the Meeting of Workmen's Compensation Committee, American College of Cardiology

I. *The committee convened* at 12 : 15 P.M. on May 28, 1959, in the Penn Room of the Benjamin Franklin Hotel, Philadelphia, Pennsylvania.

II. *The following were present,* constituting a quorum: L. H. Sigler, Chairman; G. W. H. Schepers, Acting Secretary; S. M. Horvath; J. M. Bodlander; I. R. Frank; D. Gelfand; R. Hamilton; and S. Dack. Drs. Bernstein and Kissane wrote to say they would be unable to attend.

III. *Review of the problem of the causal relationship between the performance of work and cardiovascular disease.* The following points were raised:

1. There is need for clarification of the denotation and connotation of existing legal phraseology relating to compensable heart disease.

2. No general rules can be formulated by the committee before legal concepts have themselves been standardized.

3. Existing so-called accepted criteria of compensable heart disease are not applicable to every case because of the wide range of individual variability.

4. Statistical evidence, although a guide, should not overrule the merits of the individual case. Thus the fact that there is less heart disease in physical laborers than in sedentary workers, does not yet disprove a causal relationship between physical stress and cardiac incapacitation in an individual case.

5. Too much of what is considered as authoritative evidence is essentially opinion of individuals. There is great need for precise facts.

6. Aggravation of pre-existing cardiovascular disease is as much part of the problem as is the production of new disease by stress. Indeed, as most adult hearts are abnormal to a greater or lesser degree, aggravation is probably a factor in the majority of cases in which causality appears to apply.

7. Emotional stress is probably as important or more important a consideration as physical strain. The emotional factor, mediating its effects through hormonal and autonomic mechanisms, may play a role in the period prior to the occurrence of the episode of physical exertion as well as at the time of the incident.

8. There is need for a special study of the effect of emotional stress on the coagulability of the blood. Studies such as these could probably be considerably facilitated if performed in relation to blood bank proceedings.

9. Greater administrative efforts should be made to record precise cardiological data at pre-employment, periodical, and discharge or re-employment physical examinations of industrial workers. This information would be of greater value if corporations would publish the statistical results of their studies.

10. While the committee recognizes that legalistic and sociologic factors are often the main determinants of workmen's compensation decisions, which not infrequently deviate markedly from reasonable scientific criteria, the formulation of standards by the College seems nevertheless worthwhile.

IV. *Review of the progress made in connection with the symposium on occupational aspects of coronary artery disease, which is currently under preparation by members of the committee, as reported in the minutes of the annual meeting of 1958.* It emerged that it has been the universal experience of all participants in this symposium that the tasks assigned to each member are too extensive to be dealt with expeditiously and synchronously by the members. It was resolved, therefore, that:

1. Each member to whom a topic was assigned should be accorded the rank of project leader with the right to enlist the aid of as many other members of the College as the scope of his contribution may require.

2. The essays should, in the first instance, attempt to collate existing data published in the literature, stressing the limits of normality, the extremes of abnormality on record, and the nuances of variation between these extremes.

3. Individual members who have facilities to obtain new information by virtue of their association with appropriate institutions (industry, universities) are welcome to add this material.

4. Completed reports are to be forwarded to Dr. Sigler who will arrange to have them duplicated for review by the committee prior to the next meeting.

5. In view of Dr. Eichert's death, as a result of an automobile accident, the review of the natural history of coronary artery disease has been reassigned to Dr. Frank.

V. *Proposals for new studies.* No new projects were agreed upon. The chairman will make proposals to the College President for revision of the membership.

VI. *Next meeting.* Provisionally the committee will reconvene on the occasion of the interim College meeting at Philadelphia during November, 1959.

VII. *Adjournment.* The committee adjourned at 2:00 P.M.

G. W. H. SCHEPERS, M.D., D.SC.
Acting Secretary
Committee on Workmen's Compensation
American College of Cardiology

The Query Corner

READERS are invited to submit queries on all aspects of cardiovascular diseases. Insofar as possible these will be answered in this column by competent authorities. The replies will not necessarily represent the opinions of the American College of Cardiology, the JOURNAL or any medical organization or group, unless stated. Anonymous communications and queries on postcards will not be answered. Every letter must contain the writer's name and address, but these will not be published.

Clinical Value of Measuring Quinidine Blood Levels in Quinidine Therapy of Acute and Chronic Arrhythmias

Query: What is the clinical value of measuring quinidine blood levels in quinidine therapy of acute and chronic arrhythmias?

Answer: The quinidine blood levels at which a therapeutic response is obtained usually range between 4 to 10 mg./L. However, an adequate therapeutic response may be obtained with quinidine levels below 4 mg./L. and there may be a failure to obtain a satisfactory response with levels over 15 mg./L. Toxic effects are usually obtained with the higher ranges but may occasionally be observed with quinidine levels below 4 mg./L. Particularly sensitive to quinidine effects are patients in the older age groups, those with advanced myocardial disease, and those with bundle branch block. Since procaine amide, potassium, and quinidine have a similar and synergistic effect upon the heart, quinidine should be administered with caution in patients receiving these drugs.

Quinidine levels and the electrocardiogram: Quinidine levels determine the quinidine concentration in the plasma at a specific time; the electrocardiogram depicts the effect of such levels on the heart. Since the tolerance to quinidine varies considerably in different patients, the cardiac effect of a given plasma level will also vary. The electrocardiogram is probably a better criterion for determining the effectiveness and early evidence of cardiotoxicity. It is particularly important to follow the electrocardiographic effects in patients with myocardial disease, especially in the presence of conduction disturbances. Lengthening of the Q-T interval is a sign of quinidine effect as opposed to toxicity which is manifested by (1) an increase in width of the QRS complex of more than 25 per cent or (2) when the QRS complex width becomes greater than 0.14 second in the patient with preexisting intraventricular conduction defect.

The relationship between quinidine levels and dosage: Quinidine plasma levels vary considerably in the normal subject following oral administration because of variations in absorption and elimination of the drug. Oral absorption is fairly rapid. After a single dose (for example, 0.8 gm.) the drug may be detected in the blood in one hour, reaches its peak level in two to three hours; it begins to decrease in four hours; approximately one-half is eliminated in eight hours. To maintain an adequate plasma level, multiple doses of quinidine sulfate must be administered, and since the peak level is reached in about two hours a frequent regimen is to administer five doses per day (0.3 to 0.4 gm.) or at five two-hour intervals. By this regimen a plasma level of 40 per cent of the peak is observed 12 hours after the last dose. Patients with renal disease and/or congestive failure tend to develop higher plasma levels which are maintained for longer periods.

For conversion of an ectopic rhythm it is advisable to use a relatively large dose of quinidine within a short period of time in order to obtain an effective plasma level. In 90 per cent of cases conversion occurs at plasma levels below 10 mg./L. For maintenance of quinidine effects following conversion to a normal sinus rhythm, effective plasma levels may be obtained by the use of quinidine sulfate administered 3 to 4 times a day.

Recently we have been using a long-acting preparation of quinidine in order to obviate the valleys that occur in dosage following the use of quinidine sulfate and to eliminate the night dose (Bellet, S., Finkelstein, D. and Gilmore, H.: Study of a long-acting quinidine preparation: Experience in normal subjects and in patients with myocardial abnormality. *Arch. Int. Med.*, 100: 750, 1957). We have found that quinidine

gluconate is more satisfactory in the long-acting preparations because it is ten times more soluble than quinidine sulfate. By its use a therapeutic plasma level may be obtained for approximately ten hours following the administration of a single dose.

Upon return to normal sinus rhythm patients are often subject to various influences, e.g., exercise, emotional disturbances, fever, etc., which, in the presence of auricular muscle damage, may result in a return of the ectopic rhythm, even in the presence of a so-called effective plasma level.

SAMUEL BELLET, M.D.
Philadelphia, Pa.

Oxygen by Pulmotor

Query: What is the value of oxygen (by pulmotor) in acute pulmonary and cardiovascular emergencies?

Answer: This query involves two questions, the value of oxygen and the value of the pulmotor.

Oxygen: There is no doubt of the value of oxygen in such emergencies. In impending respirocardiac failure, 100 per cent oxygen should be administered without delay. The method of administration is determined by the degree or stage, of hypoxia, i.e., depression, spasticity or flaccidity. (See *Am. J. Cardiol.* 2: 513–515, 1958.) In each stage the indications for treatment are clear and definite. These indications are met by three methods: inhalation, transpharyngeal insufflation and endotracheal insufflation, conveniently applied by a variety of gadgets.

Pulmotor: The pulmotor has been replaced by the respirator. Respirators are of two types, blow and suck (positive and negative pressure) and blow (positive pressure only). The blow type is preferred. This gadget produces its effect by carrying out the principle of "transpharyngeal insufflation." However, in the stage in which it is indicated, flaccidity, it is of little value compared to the endotracheal method. To summarize: The pulmotor, in the stage of depression, is useless; in the stage of spasticity it is useless; in the stage of flaccidity, its efficiency does not begin to equal the endotracheal technic.

PALUEL J. FLAGG, M.D.
New York, N. Y.

Book Reviews

Roentgenology of the Chest, edited by C. B. Rabin. Charles C Thomas, Springfield, Ill., 1958, pp. 504, $19.50.

Direct sponsorship of books of multiple authortype has been added to the extensive postgraduate educational program of the American College of Chest Physicians. Their intended purpose is to present the multiple facets of a field of interest from different and new points. In *Roentgenology of the Chest*, diseases of the lung, pleura, and diaphragm are admirably covered. The section on cardiovascular roentgenology, in comparison, is somewhat off balance, being assigned only 110 of a total of 504 pages. Still the general impression remains favorable, the book is well done and very informative (mostly in the noncardiac section) and is to be recommended very highly.

The pulmonary section is ably introduced by Rigler, discussing possibilities and limitations of x-ray diagnosis, followed by a lucid presentation of the normal roentgen anatomy by Medelman. The chapter on fluorography for mass screening is written by its very pioneer and its most enthusiastic advocate, Manoel de Abreu. The chapters on sectional radiography by Kane and bronchography by L. Farinas and Gomez-Zelvador are most informative. Some physiologic factors are discussed by Westermark.

Obviously in a multiauthor book it is almost impossible to attain a perfect balance between chapters. Most of them give a lucid discussion of their assigned topic. Some are indeed outstanding, such as the chapters on pulmonary mycoses, linear shadows of the lung, diffuse interstitial pulmonary fibrosis, and diseases of the diaphragm.

The cardiac section contains chapters on the x-ray appearance of the normal heart (Master, Donoso) and electro- and roentgenkymography by Dack. The historical background of angiocardiography is discussed by one of its pioneers, Lopo de Carvalho. Dotter's chapter on the acyanotic forms of congenital heart disease is brief and characterized by a complete absence of illustrations, an unusual feature in a book on radiology. The cyanotic types of congenital malformation on the other hand are discussed lucidly in a well illustrated chapter by Gasul, Squire, and Park. The most important entities are described as to radiographic, dynamic, and anatomic aspects. Venous angiocardiography is simply presented. Steinberg's chapter on diseases of the pulmonary arteries and veins is scholarly and clearly written and is a pleasure to read. The chapters on acquired diseases of the heart and pericardium are too brief to be informative.

The printing of text and illustrations is excellent, as is the format and indexing of the book.

The book is highly recommended to internist, cardiologist, and chest physicians. It is informative and up-to-date in most sections. Its shortcomings are few and not significant.

ARTHUR GRISHMAN, M.D.

The Care of the Geriatric Patient, edited by E. V. Cowdry. C. V. Mosby Company, St. Louis, 1958, pp. 438, $8.00.

This book addressed to the physician in his high calling of guide, philosopher, and friend can be recommended with certain reservations not only to physicians, medical students, and persons of the allied professions interested in aging, but also to those who should be. Since cardiovascular disease is the commonest illness in old age, the cardiologist can read this book with profit. Although the book has no specific chapter on cardiovascular disease, almost each chapter contains one or more nuggets of valuable information on the proper care of older people with cardiovascular complications.

Twenty-one contributors have written the nineteen chapters of this handy, well-bound, pocket-sized manual. The first five chapters were originally presented in 1955 as part of a symposium before the Los Angeles County Medical Association and then included in this book after revision. The chapters include material on the physician and the geriatric patient, psychologic aspects, medical aspects, mental aspects, surgical aspects, anesthesia, drugs, nutrition, dental care, genetics, nursing, hospitalization, proprietary and nonprofit nursing homes, home care, rehabilitation, geriatric training, organiza-

tions and services for older people, and geriatrics around the world.

For the physician who is faced with the problem of restoring his patient to health and full functional status in the community, the chapter "Organizations and Services for Older People" by Louis H. Ravin and Clark Tibbitts provides valuable information concerning the constellations of programs, facilities, and services offered by national, state, and community agencies and organizations.

Unfortunately the essentially high caliber of this useful and informative book is slightly marred by the obvious unevenness of style and some careless errors which closer proofreading would have undoubtedly corrected. It is hoped that future editions will eliminate these. Most of these errors appear in the chapter "Medical Aspects of Geratric Care," which is the most important chapter in a medical book for physicians. The author does not elucidate when he writes, on page 76, "Adequate hearing is necessary and should be corrected by hearing aids, properly adjusted." In the statements "The ingredient that appears to be associated with the deleterious effect of the high fat diet seems to be the unsaturated fatty acids. These are higher in some animal fats and lower in some vegetable fats and some marine oils" (p. 56), the author apparently means *saturated* rather than unsaturated fatty acids. On page 79 he recommends that the stool specimen be examined "after four days on meat and chlorophyll diet," when a *meat-free* diet is probably meant.

In this same chapter the discussion concerning hormonal factors in aging is interesting, but the failure to mention, here or elsewhere, the new orally administered drugs for the treatment of diabetes in older people—one of the greatest advances in geriatric therapeutics—is a serious oversight.

An interesting difference of opinion exists concerning weight reduction. Chapter 3 states that "weight reduction programs in the aged must be more rigorous than in the young. The caloric intake must often be extremely low to cause loss of weight." A typical 570-calorie diet is then given in Table 10. Chapter 8, on the contrary, describes the dangers of drastic weight reduction, such as accentuation of catabolic processes, and cautions "As a rule, less than 1000 calories should not be prescribed for ambulatory patients." With this statement the reviewer agrees. "Nutritional Requirements of the Aged" is one of the best chapters in the book.

The concluding chapter, "Geriatrics Around the World," provides an excellent perspective of the problem of aging outside the United States. It reminds the reader that the increase in the percentage of older people throughout the world is a result not only of our modern control over the natural processes of denudation which increases the life expectancy at birth, but also of the decline in the birth rate during the second half of the century. Frequent reference is made to De Gamles By (*The Old People's City*) in Copenhagen. Here Dr. Geill and his staff are studying blood coagulation, thrombosis, and arteriosclerosis in their elderly patients. This reviewer believes that more physicians will become interested in the problems of older people and provide better medical care for them if, like Dr. Geill, they have the opportunity of conducting research along with their medical care in the numerous institutions for the aging and chronically ill patients throughout the world.

RAYMOND HARRIS, M.D.

Abstracts

Highlights of Scientific Session

American College of Cardiology, Eighth Annual Convention*

THE ELECTROCARDIOGRAM DURING PHYSICAL EXERCISE. *Alvin Freiman, M.D., Paul Ruegsegger, M.D., Ramon Abarquez, M.D.* and *John S. LaDue, M.D. Ph.D.*, Dept. of Cardiology, Memorial Center for Cancer and Allied Diseases, New York, N. Y.

An electronic system for recording the electrocardiogram during physical exercise has been devised. This system enables the investigator to record in a continuous manner the dynamic effects of exercise on the electrocardiogram and is in contrast to other technics in which the electrocardiogram is recorded after the exercise has been terminated.

The technical recording of the electrocardiographic signal was accomplished in the following manner. Stainless steel mesh electrodes possessing great flexibility were attached to the skin at either of two locations previously chosen to give a good electrocardiographic signal with minimal muscle noise. The signal was then passed through suitable amplifiers and recorded for permanent use on tape. The taped signal was subsequently analyzed for the quality of the electrocardiographic component, for the intensity and frequency of muscle noise and for the intensity of baseline shift. On the basis of this analysis the original taped signal was then passed through suitable filters to eliminate distortion. During moderate body movements a pass band of 0.1 to 20 cps proved satisfactory while strenuous body movements required a pass band of 0.8 to 10 cps.

In the initial portion of the investigation, the electrocardiogram was recorded from a series of fifteen normal males who performed nine standardized exercises representing the most commonly used movements of the extremities and trunks. In all cases the electrocardiograms were technically satisfactory.

Following this, a group of fifteen subjects with proved cardiac disease were studied. Continuous electrocardiograms were recorded during the performance of the same nine exercises. Satisfactory tracings were again obtained in all cases, and it was possible to demonstrate on these dynamic electrocardiograms A-V blocks; intraventricular blocks, both right and left in type; arrhythmias including auricular fibrillation and premature contractions; and ST and T wave abnormalities.

Finally, a group of twenty normal subjects were studied and the feasibility of continuous recording of the electrocardiogram during walking and climbing stairs was demonstrated.

SERUM TRIGLYCERIDE AND RADIOACTIVE FAT TOLERANCE STUDIES IN CORONARY ARTERY DISEASE. *Donald Berkowitz, M.D., John J. Spitzer, M.D., David M. Sklaroff, M.D.* and *William Likoff, M.D.* Dept. of Medicine, Dept. of Physiology and Dept. of Radiology, Albert Einstein Medical Center, Northern Div. and The Hahnemann Medical College, Philadelphia, Pa.

Previous experiences with a radioactive triolein tolerance test in patients with coronary atherosclerosis have demonstrated significant deviations from the normal. These are characterized by elevated blood radioactivity levels at the peak time and twenty-four hours after ingestion of the test meal. The constancy of these results lends direct support to the premise that an abnormality in fat metabolism is present in this disease. Recently, it has been shown that this abnormality may be in the utilization of the triglyceride fraction, a major constituent of the total serum lipids.

We have attempted to determine whether or not there is any correlation between the radioactive fat tolerance and the triglyceride concentration. After a test meal of I^{131}-triolein, serial blood samples were analyzed for whole blood and lipid radioactivity and triglycerides. Normal subjects and patients with coronary artery disease (both with normal and elevated cholesterol levels) were studied.

In the fasting state, the triglyceride values were higher in the group with coronary disease, particularly when hypercholesterolemia was present. After ingestion of the tagged fat meal, hourly blood samples showed a parallel and progressive rise in the radioactivity and triglyceride concentration over a four- to eight-hour period, and then a slower decline. In those with coronary artery disease these levels were higher and more prolonged.

The present data suggest that the lipid metabolic defect in coronary atherosclerosis may be associated with a derangement in the handling of the triglyceride fraction.

THROMBOENDARTERECTOMY FOR CORONARY ARTERY DISEASE. *Charles P. Bailey, M.D., Dryden P. Morse, M.D.* and *William M. Lemmon, M.D.* Dept. of Thoracic Surgery, Baily Thoracic Clinic, Philadelphia, Pa.

Since the first attempt at coronary endarterectomy by one of us on October 29, 1956, great attention and

* May 26–29, 1959, Philadelphia, Pa.

considerable investigation has been devoted to this general approach to the problem of coronary artery disease.

Recent evidences which suggest a relative ineffectiveness of many of the currently employed operative approaches in coronary artery disease lend impetus to the concept of a more direct attack. The fact that the smaller blood vessels of the coronary arterial system characteristically remain patent, even after complete occlusion has taken place in the major arterial trunks, suggest that restoration of the lumen of these latter vessels might bring about an effective degree of "revascularization" of the ischemic myocardium. Obviously, however, in complete infarction or extensive fibrosis of the myocardium, restoration of the vascular lumens would not be of value. Therefore, one might well presume that the value of thromboendarterectomy would be most apparent in those cases in which complete occlusion had not yet taken place in the major coronary trunks; or if it had taken place, in those of recent origin (not over six hours). It is possible that some benefit might be obtained even at a later date than this if one were willing to accept a higher degree of vascularization of the peri-infarctional area for the more limited benefit which this might provide.

Studies with thrombosis induced in animals suggest that after the passage of a year or more, the organization which takes place renders effective surgical separation of the thrombus from the vessel wall impractical. Clinically, the same thing has been experienced in removing old thrombi from the popliteal and internal carotid arteries. Therefore, it is obvious that for benefit to be achieved following occlusion, necessarily the procedure should be performed within a year or so.

In partial occlusions which frequently precede and forebode the development of a complete occlusion, the operation of endarterectomy or thromboendarterectomy should be ideal.

The acute surgical removal of a thrombus along with the damaged intima of a vessel following acute infarction may be considered as comparable to the performance of a similar operation for acute thrombosis of the femoral artery. It is believed that this will be the method of treatment of acute coronary occlusion in the future. From experimental studies it would seem that at least four hours exist after the onset of an acute occlusion before infarction has become irreversible during which successful thromboendarterectomy might be anticipated. The precarious condition of the patient at the time of induction of anesthesia and surgery does not preclude this attempt because the heart-lung machine may be adapted for the support of the circulation during this period and during the definitive surgical procedure. Postoperatively the patient's clinical improvement should permit his successful recovery.

Fibrinolysin, used judiciously before and after the performance of thromboendarterectomy, has been proved, both in the laboratory and clinically, capable of preventing postoperative thrombosis at the site of vascular traumatization.

RELATIONSHIP BETWEEN ELECTRICAL AND MECHANICAL ACTIVATION. *Philip Samet, M.D., Robert S. Litwak, M.D.* and *William H. Bernstein, M.D.* Cardio-Pulmonary Lab. and Dept. of Medicine, Mt. Sinai Hospital, Miami Beach, Fla., and Div. of Cardiology, Dept. of Medicine and Surgery, Univ. of Miami School of Medicine, Coral Gables, Fla. and Jackson Memorial Hospital, Miami, Fla.

Previous studies employing electrokymography have failed to demonstrate delayed onset of ejection in either great vessel in the presence of bundle branch block or ventricular premature beats in man. The development of combined right and left heart catheterization has afforded a technic for the simultaneous recording of right and left ventricular pressure pulses during sinus rhythm or atrial fibrillation and during ventricular premature beats. Significant delay in the onset of mechanical isometric contraction in the ventricle on the side opposite to the site of origin of the ventricular premature beat was not demonstrated during the study in man. The problem was, therefore, taken into the experimental laboratory. Simultaneous right and left ventricular pressure pulses were recorded in the dog during the course of over 400 mechanically induced ventricular premature beats and during the course of 2,500 ventricular premature beats with widened aberrant QRS complexes induced by the administration of digitalis. The results demonstrated that the delay in mechanical contraction of the side opposite to the site of origin of the ventricular premature beat was only minimal and of questionable physiologic significance. The delay was far less than expected on theoretic grounds. Right and left bundle branch block and complete heart block were also produced in a number of experimental dogs to note whether or not there would be delay in onset of ventricular contraction on the ipsilateral side in the case of bundle branch block. Again, the delays were found to be minimal and of questionable physiologic significance. These varied studies, therefore, demonstrate that asynchronous electrical activation of the ventricles does not necessarily result in significant delay of onset of mechanical contraction as would be expected on theoretic grounds.

A SIMPLE METHOD OF RECORDING AND INTERPRETING THE SPATIAL ELECTRICAL FIELDS OF THE HEART. *Julien H. Isaacs, M.D.* and *J. Louis Freibrun, M.D.* Dept. of Cardiology, Univ. of Southern California School of Medicine, Los Angeles, Calif.

Spatial vectors representing the electrical fields of the heart can be recorded as potential changes on standard office electrocardiographic apparatus by (1) utilizing any vectorcardiographic lead reference system or electrode placements; (2) obtaining vector potentials from the leads representing the spatial coordinate body axes, X (left-right), Y (vertical) and Z (anteroposterior); and (3) separately recording each of these X, Y and Z vector potentials on a single electrocardiographic lead. This method eliminates many of the technical disadvantages inherent in vector loop recording. Special skills and equipment are unnecessary. Recording is simple and requires but a few minutes. Clear tracings are obtained without effort; measurement of magnitude, direction and timing are simplified, and arrhythmias can be readily studied.

The recorded vector potentials, X, Y and Z are analyzed with a simplified method of interpretation that can be applied to any vectorcardiographic lead reference system. The fundamental principles of interpretation are based on timing, direction and relative magnitudes of the instantaneous activation and recovery potentials. Pathologic states are detected, identified and localized

by specific changes in timing, direction and magnitude of these activation and recovery potentials. This method retains all the experience and advantages of recent and past studies of electrocardiography and vectorcardiography and, in addition, simplified recording, orientation, and interpretation. Clinical interpretation and derivation of vector loops are possible by inspection.

DETECTION OF PULMONIC AND TRICUSPID VALVULAR REGURGITATION WITH AN INDICATOR-DILUTION TECHNIC. *N. Perryman Collins, M.D., Eugene Braunwald, M.D.* and *Andrew G. Morrow, M.D.* Clinic of Surgery, National Heart Institute, Bethesda, Md.

The definite diagnosis of pulmonic and of tricuspid valvular regurgitation may frequently be difficult or even impossible with established diagnostic methods. A technic has been developed which permits the demonstration of blood traversing these valves in a reverse direction and an estimation of the magnitude of regurgitant flow. When the competency of the pulmonic valve was studied, a modified double-lumen cardiac catheter was positioned so that the distal lumen opened into the pulmonary artery while the proximal lumen opened into the right ventricle. When tricuspid valve function was examined, the distal lumen was in the right ventricle while the proximal lumen was in the right atrium. Cardio-green dye was injected through the distal opening of the catheter and an indicator-dilution curve was recorded with blood sampled from the proximal opening. With a competent valve, either no indicator, or only a minimal quantity, could be detected in the proximal chamber immediately after injection. In the presence of valvular regurgitation, substantial amounts of indicator appeared in the proximal chamber immediately after injection. The regurgitant fraction was calculated as the ratio of the product of the build-up time and peak concentration of the regurgitant curve to that of the recirculation curve.

Regurgitation was present in four of twenty-one patients in whom the pulmonic valve was examined, with regurgitant fractions ranging from 17 to 53 per cent. Tricuspid regurgitation could be proved in five of thirteen patients studied; the regurgitant fractions were 22 to 65 per cent. The method described appears to be reliable, simple to apply during the course of right heart catheterization, and of clinical value in the study of patients with valvular heart disease.

HEART DISEASE, EXERCISE AND CARDIAC OUTPUT. *Sylvan L. Weinberg, M.D., G. Richard Grove, Ph.D.* and *Robert E. Zipf, M.D.* Dept. of Research, Miami Valley Hospital, Dayton, Ohio.

Patients with heart disease were subjected to a standard Master's two-step test and the cardiac output determined by external monitoring over the heart of intravenously injected radioiodinated serum albumin (RISA). The group included instances of arteriosclerotic and hypertensive cardiovascular disease with and without congestive heart failure and remote myocardial infarction. Serial cardiac output determinations were made at rest, immediately, four and a half and eight minutes after exercise. The expected cardiac output values had been established previously in normal patients. Electrocardiographic monitoring was carried out at the time of each cardiac output determination. The cardiac output response curve varied from normal patterns to grossly disordered ones. Abnormal output patterns were not necessarily accompanied by positive electrocardiographic changes after exercise. Principal abnormal output findings included low resting values, less than normal rise after exercise and failure of the eight-minute output after exercise to return to the resting level. In normal persons the eight-minute cardiac output after exercise had returned to the resting or a slightly lower level. In several instances, an elevated eight-minute output value was the only abnormality detected.

It is believed that the method of cardiac output used in this study has achieved clinical applicability. This view is based on demonstrated simplicity, accuracy and precision of the technic. The pattern of serial cardiac output studies in response to exercise is helpful in defining the extent of a disease process in a given individual in terms of cardiac dynamics and in estimating response to therapy.

CLINICAL OBSERVATIONS ON A NEW CORONARY VASODILATOR (WIN 5494). *Raymond Harris, M.D.* Dept. of Cardiology, St. Peter's Hospital, Albany, N. Y.

The newly synthesized compound, 3-dimethylamino-1,1,2-tris-(4-methoxy-phenyl)-1-propene hydrochloride, is a unique type of coronary vasodilator drug with a pattern of activity similar to that of the nitrites. On the basis of promising laboratory experimental results a clinical trial of this drug was inaugurated in patients with coronary artery disease who suffered from angina pectoris, myocardial infarction, or some type of arrhythmia, usually ventricular premature contractions.

Twenty of thirty-two patients with angina pectoris showed marked and definite improvement after being placed on Win 5494; nine had moderate improvement; three had equivocal or no objective improvement. These ratings were made after comparison of the patients' responses to similar placeboes, other coronary vasodilators and Win 5494.

Since animal experiments indicated this drug had some antiarrhythmic effect in addition to its coronary vasodilator action, it was administered to eighteen patients with ventricular premature contractions who were under constant observation, including frequent electrocardiograms. The oral administration of 50 mg. of the drug either eliminated entirely, or reduced appreciably, the ventricular premature contractions found before administration of the drug in sixteen of eighteen patients studied repeatedly. It abolished paroxysmal atrial tachycardia in one patient which returned when the drug was stopped. One of six patients with atrial fibrillation showed reversion to sinus rhythm during the use of this drug. In another patient with atrial fibrillation paroxysmal atrial tachycardia developed.

The drug was also administered to patients with acute myocardial infarction without adverse effects. No serious side-effects were noted.

The results of this study indicate that Win 5494 is a promising drug for the treatment of angina pectoris and certain arrythmias, especially ventricular premature contractions.

IMPENDING MYOCARDIAL INFARCTION; RECOGNITION AND MANAGEMENT. *R. E. Beamish, M.D.* and *V. Marie Storrie, M.D.* Winnipeg, Canada.

Not infrequently, myocardial infarction is preceded by

premonitory pain. This symptom usually begins rather abruptly suggesting a thrombotic genesis. It seems reasonable to attempt early recognition of such patients in order to administer anticoagulants in the hope of preventing subsequent infarction.

From a study of over 100 patients, and of the literature, it is concluded that there are three groups of patients in which infarction may be considered imminent. These groups are: (1) patients who have never experienced cardiac pain who suddenly experience it for the first time; such pain may occur at rest or take the form of rapidly intensifying angina of effort; (2) patients who have had angina of effort for months or years in whom the symptom abruptly intensifies; (3) patients with previous infarction who have been pain-free for months or years, and then begin having pain again. Clinical and cardiographic features of these groups will be described.

Of the first 100 patients diagnosed as having an impending infarction, eighty-five received prompt anticoagulant therapy. The remaining fifteen patients either declined treatment or were unable to be admitted to a hospital for treatment. This latter group serve as "controls." The outcome in these two groups of patients is considered under three phases: (1) the first six weeks (acute phase), (2) six weeks to six months (chronic phase) and (3) after six months (late phase).

Of the eighty-five patients who received anticoagulants, only two had infarcts within six weeks and neither of these was fatal. Three more had infarcts within six months, two of them being fatal. Ten had infarcts, seven being fatal, after six months. In contrast, in the fifteen "controls" fourteen had infarcts, eleven of them being fatal. Nine of these deaths occurred within the first six weeks and two within six months.

The number of patients diagnosed as having an impending infarction can be increased by a program of patient-education. This concept will be briefly described.

INTRAVENOUS INJECTION OF HYALURONIDASE IN ACUTE MYOCARDIAL INFARCTION: PRELIMINARY REPORT OF CLINICAL AND EXPERIMENTAL OBSERVATIONS. *Henry A. Zimmerman, M.D.* and *Jorge Martins de Oliveira, M.D.* St. Vincent Charity Hospital, Cleveland, Ohio.

Observations on the use of intravenous hyaluronidase were made in dogs in which acute coronary insufficiency was produced by ligating branches of the coronary arterial tree, and in seven patients with acute myocardial infarction. Particular attention was given to the electrocardiographic changes observed after the injection of the enzyme.

The most impressive finding in both man and dogs has been the rapid improvement observed in the ST segment. In six of the patients the ST segment shifted toward normal in a few hours after the administration of hyaluronidase. In two cases rapid improvement in disturbances of conduction was noted. In dogs, marked decrease in the current of injury was observed within a few minutes after the intravenous administration of the enzyme.

Hyaluronidase is thought to act by reducing edema in the damaged myocardial area, thus decreasing the injury to the cardiac fibers during the critical first stage that follows acute coronary occlusion; it also facilitates the beneficial action of a possible existent collateral circulation. The mechanism through which the edema is reduced is not as yet well established. It is thought that hyaluronidase may spread the edematous process and this would facilitate capillary reabsorption.

THE PROBLEM OF CEREBRAL ANGIOSPASM. *Eliot Corday, M.D.* and *Sanford F. Rothenberg, M.D.* Cedars of Lebanon Hospital, Los Angeles, Calif.

The problem of cerebral angiospasm still remains unsettled. In order to solve this problem, we have photographed the arteries of the base of the brain and demonstrated that they may respond to external stimuli such as trauma, topical application of adrenalin, etc. In addition, it was noted that the arteries respond passively to changes in the systemic blood pressure.

Studies of cerebral artery flow were made which demonstrated that the control of cerebral blood flow was mainly in the extracranial portion of the carotid and vertebral arteries. Intracranial angiospasm may account for ischemic cerebral phenomena only if trauma or humoral substances are present.

APICAL DIASTOLIC AND PRESYSTOLIC MURMURS OF PROVED FUNCTIONAL NATURE. *Aldo A. Luisada, M.D., Jan Szatkowski, M.D., Mario R. Testelli, M.D.* and *Jesus Bendezu-Prieto, M.D.* Div. of Cardiology, The Chicago Medical School, Chicago, Ill.

We have collected a series of thirty-two clinical cases in which a diastolic or presystolic murmur simulating that of mitral stenosis was recorded in the phonocardiogram. The functional nature of the murmur was proved in nine by autopsy; in ten, by right heart catheterization (pulmonic stenosis or septal defect); and in thirteen by left heart catheterization, which revealed the absence of a mitral gradient.

A clinical error was made in most cases, except those in which an entirely different clinical picture was present (congenital heart disease, recent myocardial infarct).

The phonocardiogram was correctly interpreted in most cases. However, in five cases the tracing was interpreted as being consistent with mitral stenosis. In two of them, a "dynamically insignificant" mitral stenosis was suspected, although *not proved*. In the other three, no stenosis whatsoever was finally demonstrated.

A case in which the murmur was probably due to acute rheumatic carditis is discussed in detail and repeated tracings are presented.

The various causes which can lead to a clinical error are listed, and it is shown that, in many of these cases, a phonocardiogram permits to exclude mitral stenosis. It is further shown that, in a few exceptions, even the phonocardiogram can be misinterpreted. Left heart catheterization often reveals the absence of a mitral block, thus reducing the doubtful cases (dynamically insignificant lesion) to a minority.

LOW FREQUENCY TRACINGS OF THE PRECORDIUM: TECHNICAL COMPARISON OF VARIOUS SYSTEMS. *Leslie M. Rosa, M.D.* Div. of Cardiology, The Chicago Medical School, Chicago, Ill.

The configuration of precordial tracings and the time relationship between the waves of the latter and those of ballistocardiograms were studied. Common basic patterns of precordial displacement, velocity

and acceleration tracings, taken with different pickups in different frequency ranges, are described.

The oscillatory pattern depends on the physical characteristics of the pickup-amplifier-filter system. The rest being equal, the filter band determines the configuration of the precordial tracing.

The precordial *displacement* oscillatory pattern in the range 0.01–100 c.p.s. is identical with the reverse picture of the ULF *displacement* ballistocardiogram.

The precordial *displacement* oscillatory pattern in the 0.01–50 c.p.s. range is similar to the ultra short wave displacement radiocardiogram, an inertialess, contactless method (Rosa). Thus, with different low pass filters, the precordial tracing loses its similarity to the ULF displacement ballistocardiogram. The precordial *displacement* tracing in the 12–20 c.p.s. range resembles the direct body acceleration ballistocardiogram; in the 5–25 c.p.s. band, it corresponds to the ULF *acceleration* (lateral) BCG.

There is practically no difference between the pattern of a precordial *velocity* tracing in the 0.01–50 c.p.s. band and that of the ULF ECG *velocity* tracing.

The precordial *acceleration* pattern in the 0.01–50 c.p.s. range is similar to the high frequency ballistocardiogram, whereas, in the 5–25 c.p.s. range, it corresponds to the ULF acceleration BCG.

The vibrations of the precordial tracing have been designated in conformity with the proposals of the American Heart Association Committee on Ballistocardiographic Terminology. Measurements on time relationships between precordial waves and the waves of the ECG gave similar results to those found by Scarborough and his group in ballistocardiograms.

The time coincidences of precordial waves with those of other cardiovascular tracings have been demonstrated and discussed.

The pulsatory vibrations of the precordium—as far as oscillatory pattern and timing is concerned—are similar to ballistocardiographic vibrations of the total body mass. The similarity to tracings recorded by different BCG technics is strictly limited to defined frequency ranges.

EXPERIMENTAL ROENTGENOLOGIC VISUALIZATION OF NORMAL AND ABNORMAL CORONARY ARTERIES. *William H. Sewell, M.D., Wade H. Shuford, M.D.* and *Pablo A. Davalos, M.D.* Div. of Thoracic Surgery of Dept. of Surgery, and Dept. of Radiology, Emory Univ. School of Medicine, and Grady Memorial Hospital, Atlanta, Ga.

This project was designed to develop a method in dogs permitting more consistent roentgenologic visualization of the smaller branches of the distal coronary arterial tree, and to evaluate the reliability and safety of the method.

Several technics have been tested, wherein contrast media was injected into the ascending aorta. Studies were made with reduction of the cardiac output by inflation of plastic bags in the venae cavae or in the ascending aorta. However, temporary cardiac asystole caused by acetylcholine was found to be the most satisfactory.

Forty-two arrests were produced in sixteen normal dogs, all of which spontaneously resumed normal rhythm after fifteen to twenty seconds. P waves sometimes returned before the QRS complex, but there were no other ventricular or auricular arrhythmias.

Twenty-three arrests were produced in nine abnormal dogs three days to four months after complete or partial surgical occlusion of either the main left coronary or anterior descending coronary artery. All hearts restarted without serious arrhythmia except one, in which ventricular fibrillation developed. Hypaque (85 per cent) was used as the contrast material.

In fifty-five consecutive arteriograms the coronary arteries were outlined for several successive branchings and vessels as small as 300 microns in diameter were consistently filled. Arteriograms of the abnormal heart showed clearly in all cases the point of partial or complete arterial occlusion. Collateral circulation was demonstrated in hearts having obstructive arterial lesions.

Announcements

International Symposium on Cardiology in Aviation

School of Aviation Medicine, Air University
Brooks Air Force Base
San Antonio, Texas

November 12–13, 1959

Sponsors:
Aerospace Medical Association.
Air Line Medical Directors' Association.
School of Aviation Medicine, Air University.

Thursday, November 12, 1959

Welcome and Introduction.
OTIS O. BENSON, JR., MAJ. GEN.
History of Cardiology in Aviation.
ASHTON GRAYBIEL, CAPT.

I. Influence of Aerospace Flight on the Normal Cardiovascular System—Stresses and Effects.
LAWRENCE E. LAMB, M.D.

II. Cardiovascular Techniques Used for Flying Personnel.
HOWARD G. BURCHELL, M.D., Presiding.

Phonocardiography.
E. GREY DIMOND, M.D.
Ballistocardiography.
JACK SMITH, M.D.
Vectorcardiography.
LAWRENCE E. LAMB, M.D.
Telemetry.
NORMAN L. BARR, CAPT.

III. Cardiovascular Disorders in Aircrew Personnel.
M. S. WHITE, BRIG. GEN., Presiding.

Pathological Findings in the Cardiovascular System of Pilots.
FRANK TOWNSEND, COL.
The Cardiovascular System of the Aging Pilot.
OTIS SCHREUDER, BRIG. GEN.
The Problem of Elevated Blood Pressure or Hypertension in the Pilot.
LAWRENCE E. LAMB, M.D.
Prognosis in Cardiovascular Findings, Based on Life Insurance Statistics.
HARRY E. UNGERLEIDER, M.D.
The Problem of Unexplained Loss of Consciousness in Flying Personnel.
GEORGE DERMKSIAN, M.D.

Panel Discussion on Cardiovascular Diseases in the Flier.
LAWRENCE E. LAMB, M.D., Moderator.

Topics:
Aviation Aspects of:
Arteriosclerotic Heart Disease
Hypertensive Cardiovascular Disease
Congenital Heart Disease
Rheumatic Heart Disease
Pericarditis
Panel Members:
HOWARD B. BURCHELL, M.D.
ROBERT GRANT, M.D.
ASHTON GRAYBIEL, CAPT.
ARCHIE A. HOFFMAN, COL.
S. W. HOOBLER, M.D.
GEORGE W. MANNING, M.D.
F. A. L. MATHEWSON, M.D.
JACK SMITH, M.D.

The National Program for Study of Cardiovascular Disease.
LEROY E. BURNEY, SURG. GEN.
U. S. Public Health Service

Friday, November 13, 1959.

IV. Electrocardiographic Studies of Flying Personnel.
HERBERT W. COONE, COL., Presiding.

U. S. Air Force Survey of 72,000 Electrocardiograms.
Part 1: Normal Values and Incidence of Pathology.
KEITH H. AVERILL, M.D.
U. S. Navy Experiences in Electrocardiographic Evaluation.
ASHTON GRAYBIEL, CAPT.
The Royal Canadian Air Force Experiences in Electrocardiographic Evaluation.
GEORGE W. MANNING, M.D.

Electrocardiographic Studies of Flying Personnel (*Contd.*)
G. EARL WIGHT, M.D., Presiding.

U. S. Air Force Survey of 72,000 Electrocardiograms.
Part 2: Supraventricular Rhythms.
KEITH H. AVERILL, M.D.
Part 3: Ventricular Rhythms.

ROLAND G. HISS, CAPT.
Part 4: Atrioventricular Block.
ROBERT L. JOHNSON, MAJ.

Electrocardiographic Studies of Flying Personnel (*Contd.*)
ARCHIE A. HOFFMAN, COL., Presiding.

Part 5: The Wolff-Parkinson-White Syndrome.
KEITH H. AVERILL, M.D.
Part 6: Complete Right Bundle Branch Block.
ROBERT L. JOHNSON, MAJ.
Part 7: Complete Left Bundle Branch Block.
KELVIN D. KABLE, CAPT.

Electrocardiographic Studies of Flying Personnel (*Contd.*)
LUDWIG G. LEDERER, M.D., Presiding.

Part 8: T Wave Variations.
ROLAND G. HISS, CAPT.
Part 9: Myocardial Infarction
GEORGE B. SMITH, JR., MAJ.

Electrocardiographic Interpretation in Stress Tolerance Test
E. GREY DIMOND, M.D., Monitor.
GEORGE W. MANNING, M.D.
ASHTON GRAYBIEL, CAPT.

V. Limits of Cardiovascular Normality for Flying—A Panel Discussion.
FREDERICK S. SPIEGEL, LT. COL., Presiding.

Panel Members:
ASHTON GRAYBIEL, CAPT.
LAWRENCE E. LAMB, M.D.
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Concluding Remarks
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